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AN OPERATIVE TREATMENT FOR CERTAIN CASES OF MENINGOCELE (OR ENCEPHALOCELE) INTO THE ORBIT *

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Pulsating exophthalmos may arise from: (1) arteriovenous aneurysms of the brain, orbit or cavernous sinus; (2) arterial and arteriovenous aneurysms of the orbit and (3) defective roof of the orbit. The treatment for pulsating exophthalmos, therefore, is dependent on and directed to the underlying cause. In the case here reported (fig. 1), the pulsating exophthalmos was due to an orbital meningocele of congenital origin. It would perhaps be more correct to speak of congenital absence of the roof of the orbit, for doubtless this defect was primary, the cerebral meningocele following in its wake as a necessary mechanical sequel. The differential diagnosis of the cause of the pulsating exophthalmos was based on the positive roentgenologic evidence that the roof of the orbit was in large part missing on the affected side (figs. 2 and 3). The explanation of the exophthalmos was that the unopposed pressure of the brain or the meningeal contents was gradually forcing the orbital contents outward (fig. 4). The pulsation was due to the direct transmission of the pulsation of the brain to the eyeball.

For such a cause the obvious treatment is reconstruction of the orbital roof, preferably by an autotransplant of bone. In this patient, a measured graft was taken from the outer table of the skull, giving much the same slight curvature that obtains in this portion of the normal orbital roof.

The approach for this exposure was through a small anteriorly placed bone flap, extending from the orbital ridge to the anterior hair line or slightly beyond (fig. 5). This approach is used as a routine for the operative attack on hypophyseal tumors and is suitable for all explorations of the roof of the orbit. A few years ago a much more extensive cranioplasty [1] was advocated as an approach to prechiasmal tumors involving the optic nerves and extending into the orbit, or for tumors arising in the orbit and extending into the cranial chamber.

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* Submitted for publication, April 25, 1929.

* From the Johns Hopkins University and Hospital.

1. Dandy, W. E.: Prechiasmal Intracranial Tumors of the Optic Nerves, Am. J. Ophth. **5**:169 (March) 1922.

Fig. 1.—Patient with orbital meningocele (before operation).

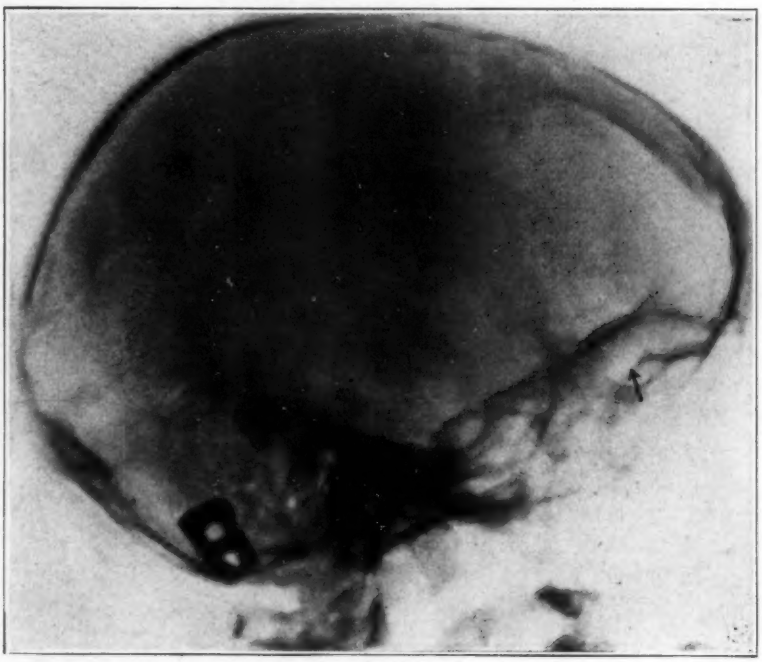

Fig. 2.—Lateral view of the skull. The arrow points to the posterior end of the orbital roof (defective) on the left side. The normal orbital roof on the right side lies just above. One can also see the greatly thinned bone over the entire frontal region, doubtless a congenital defect.

Experience, however, has shown that in most instances the restricted exposure serves equally well and has the other advantages of being a less formidable procedure and of reducing the incidence of postoperative extradural hemorrhage. Had an encephalocele been encountered instead of a meningocele, its removal should not have been difficult owing to the ample room provided by the operative approach.

The bone transplant was obtained along the posterior line of incision and back of the hair line. The entire thickness of bone was removed, then split by chiseling through the diploe. The outer table was used for the graft.

Fig. 3.—Anteroposterior view of the skull. The two arrows on the left indicate the normal roof of the superior orbital fissure. This is absent in the right side (left eye).

A search of the literature on meningocele, encephalocele and congenital malformations of the orbit indicates that the lesion is rare. No record was found of an operative procedure directed toward a cure.

REPORT OF A CASE

History.—A bright girl, aged 16, was referred by Dr. Alan C. Woods because of a unilateral pulsating exophthalmos due to a defect in the roof of the orbit. She had previously been seen by Dr. Arnold Knapp of New York. From a series of x-ray pictures, Dr. Knapp had first made the diagnosis of congenital absence of the left roof of the orbit.

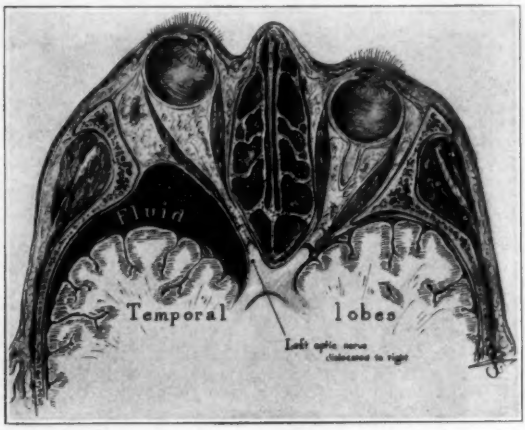

Fig. 4.—Sketch showing operative observations. The bed of fluid represents the meningocele which is pushing the orbital contents forward, downward and inward.

Fig. 5.—Photograph taken two weeks after the operation. The exophthalmos is still present due to the edema. The operative scar is entirely beneath the hair line.

Soon after the birth of the child the mother noticed that the left eye protruded slightly and was a little lower than the right. Slowly the eye became more prominent, and a definite internal strabismus developed. One year before examination the exophthalmos seemed to increase rather abruptly and at the same time the vision was affected. There had been no headaches or other symptoms.

Examination.—Aside from the local condition and related functions, a minor congenital deformation of the ear and marked thinning of the skull in the frontal region (lateral view), the physical and neurologic examinations revealed nothing. Her general condition was excellent.

The report of Dr. Woods' examination was as follows: "Exophthalmometer readings: Right 19 mm., left 24 mm. The best vision on the right is 20/30 +; on the left 20/100. There is a slight concentric contraction of the left visual fields though the change is not great (fig. 6). In addition to the exophthalmos, the left eye deviates downward and inward. A fine vertical nystagmus is present. The optic disc is normal in outline; the physiological cup is a little blurred; the disc is

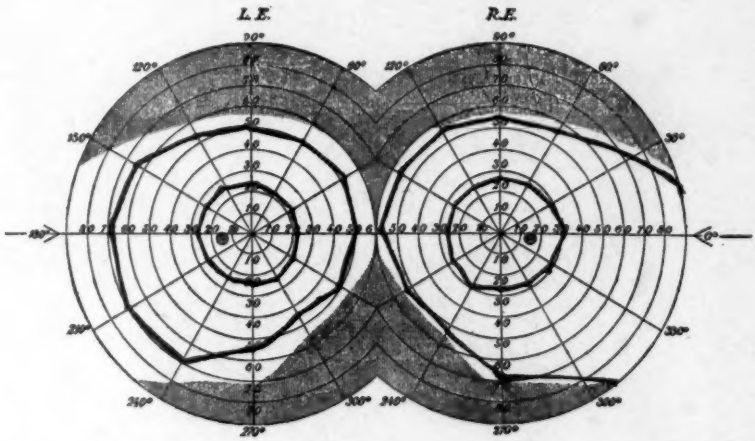

Fig. 6.—Visual fields of patient before operation, May 28, 1927. The best vision on the right was 20/30, that on the left, 20/100.

flat with slight overfilling and rather marked tortuosity of the veins. The pupils are equal and react normally to light and accommodation."

On looking at the eye from a lateral position, a pulsation, synchronous with the pulse, was easily seen. It was also palpable, but there was no thrill. No murmur was audible. X-ray pictures of the skull taken laterally and anteroposteriorly (both stereoscopic) showed that the posterior half of the roof of the orbit was missing. There was a small shelf internally and externally. All of these observations had previously been made by Dr. Knapp.

Operation.—On July 15, 1927, a transplant of bone from the outer table of the skull (fig. 7) was bridged across the roof of the orbit. The curvature of this bone was essentially the same as the arch of the orbital roof. Moreover, the external and internal margins of the orbit were adequate to give a base on which the graft could rest and to which it should ultimately unite. A bed was chiseled from the external rim of the orbital roof where it joins the side of the skull, and into this notch the outer margin of the transplant fitted accurately and firmly. The graft was passed through a tunnel between the dura and the orbital contents

(fig. 8). The inner margin of the transplant rested on the inner rim of the orbital plate, from which the dura and periosteum had been scraped to permit the transplant to lie in apposition with bare bone. The ends of the graft fitted so accurately that it was not necessary to wire the graft in place. The new superior orbital fissure was probably a little wider than normal but the difference was not great.

Other Details of the Operation and Abnormal Observations.—The outer surface of the brain was covered with large pools of fluid in the subarachnoid space; the leptomeninges were opaque and greatly thickened. The release of this fluid yielded adequate room to permit elevation of the brain without producing trauma. It was not necessary to puncture the lateral ventricle or to withdraw spinal fluid by lumbar puncture, as is the custom when intracranial room is at a premium.

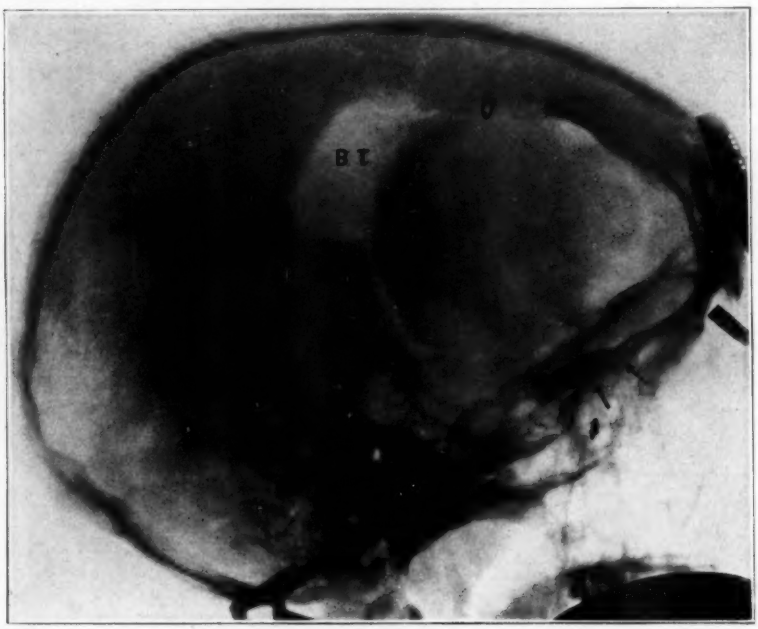

Fig. 7.—Lateral view of the skull taken eighteen months after the operation. The two arrows point to the shadow of dense bone which represents the transplant of bone. The normal orbital roof of the right side is seen just above. The three clusters of silver clips are on vessels which were ligated and divided as they crossed from the orbit to the temporal bone. The clear area *B.T.* is the defect from which the bone transplant was taken.

On retraction of the frontal lobe the defect in the posterior two thirds of the orbital roof showed precisely as in the x-ray picture. The bony defect was everywhere covered by dura which was firmly attached to the eyeball (fig. 9). The dura extended backward with a gradual decline to and across the floor of the middle fossa. An isolated small fragment of bone lay between the dura and the eyeball.

The tip of the temporal lobe was covered by tremendously dilated subarachnoid spaces (fig. 4), with opaque membranes—changes not unlike those on the

surface of the brain, except in degree. Superficial examination might well give the impression that the tip of the temporal lobe, which was entirely hidden from view, was covered by an actual cyst. These large subarachnoid spaces were continuous through the sylvian fossa with similar spaces over the frontal and parietal lobes. The maximum depth of fluid was about 2.5 cm. The intact temporal lobe lay beneath the bed of fluid.

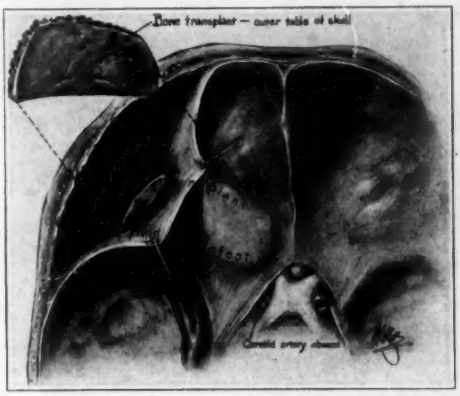

Fig. 8.—Drawing to show the implantation of the bone through a subdural tunnel. The bone rested internally and externally on solid bone.

Fig. 9.—Drawing of the field of operation, showing the defect in the posterior part of the orbit. The large extradural vein doubtless represents the cavernous sinus. The internal carotid artery was absent.

Three small veins crossed from the external surface of the temporal lobe to the orbital tissue piercing the dura en route. They were doubly ligated with silver clips and divided. With these veins out of the way and the bed of fluid evacuated, the view of the entire anterior and middle fossa was unobstructed. A tremendous anomalous vein lay along the inner side of the middle fossa and outside the dura (fig. 9). It was formed by the confluence of two large veins—

doubtless the superior and the inferior orbital veins—which also pierced the dura covering the orbital contents. It doubtless represented an extradural cavernous sinus. Posteriorly, the ultimate destination of the vein could not be traced.

The left optic nerve was bent to the right, due to the pressure of the large cup of fluid on the tip of the temporal lobe. Search was made for the carotid artery which always stands out conspicuously alongside the optic nerve, but it could not be found (fig. 9). It is interesting that both the internal carotid artery and the cavernous sinus were absent. Knowing that the carotid artery in its transit through the skull passes through the cavernous sinus, I was led to wonder if the absence of the cavernous sinus might not lead to the loss of the internal carotid artery. No attempt was made to disclose the other nerves (III, IV and VI) entering the orbit. The anterior clinoid process on the left side was absent.

Postoperative Course.—Immediately after the operation there was perhaps a tiny exophthalmos of the affected eye, though the difference between the position of the two eyes was slight. During the succeeding two or three weeks there was some protrusion, doubtless from edema of the orbital tissues (fig. 5). This gradually subsided, and the eye has since been in almost normal position in the anteroposterior direction.

The following observations on the eye were recorded by Dr. Woods, subsequent to the cranial operation:

Sept. 27, 1927 (Ten Weeks After the Orbital Transplant): A marked edema of the orbit and further protrusion of the eye which occurred after the operation had disappeared. To protect the eye, a median tarsorrhaphy was done (by Dr. Woods). The degree of exophthalmos was 19 mm. right and 21 mm. left. The eye still showed 10 per cent of internal strabismus with 5 per cent of deviation downward. The media and fundus were unchanged. Slight nystagmus still persisted.

Nov. 27, 1927 (Nineteen Weeks After the Orbital Transplant): There was no relative exophthalmos. Exophthalmometer readings were 19 mm. for both eyes. The internal strabismus of the left eye measured 25 degrees; the downward deviation was 5 per cent. Corrected vision remained 20/30 + right, and 20/100 left.

Operation on the External Rectus Muscle by Dr. Woods.—The extra-ocular palsy was, of course, not changed by the orbital operation. Seventeen months later (Dec. 6, 1928), Dr. Woods operated to correct the internal strabismus and vertical deviation. His note on the operation was as follows:

Resection and advancement of the external rectus muscle on the left side was performed. The upper fibers of the tendon were advanced slightly more than the lower fibers in order to get some correction of the vertical deviation. The eye healed without complications, and the deep sutures were removed a month later. The eyes were entirely straight so far as lateral deviation was concerned, and there was no vertical deviation downward. The left eye, however, appeared to be on a lower level than the right, being about 2 mm. lower in the orbit. For this condition I do not think anything could possibly be done, nor do I think anything should be attempted.

Final Results.—The appearance of the eye at the time of writing can be seen in figure 10. A slight downward displacement of the eyeball still remained. There was no exophthalmos or pulsation of the eyeball. The eyes focused well. The extra-ocular movements were nearly perfect five months later. There was no diplopia. Figure 11 shows the graft to be apparently unchanged.

Fig. 10.—The appearance of the patient twenty-one months after operation. There is no exophthalmos and no pulsation of the eyeball.

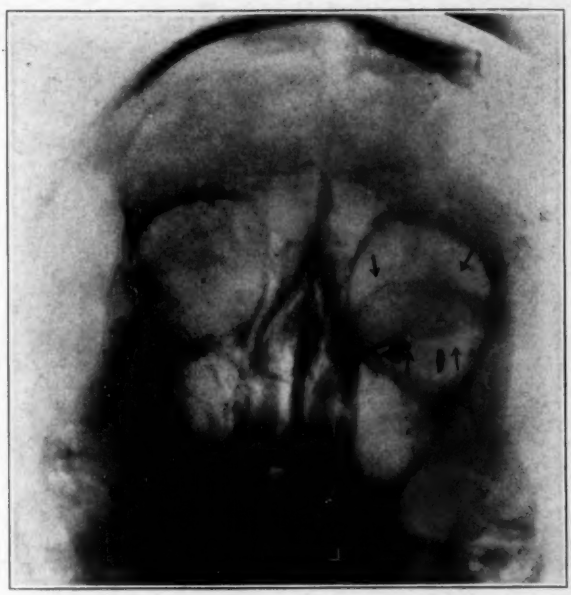

Fig. 11.—Anteroposterior view of the skull taken eighteen months after operation. The arrows point to the upper and lower margins of the transplant of bone.

SUMMARY

1. A case of pulsating exophthalmos, due to congenital absence of the orbital roof, is reported.

2. The pulsating exophthalmos was cured by covering the orbital defect with a transplant of bone taken from the outer table of the skull.

3. Other associated congenital deformations were encountered at operation: (1) a meningocele, (2) a large extradural vein which apparently replaced the cavernous sinus and (3) an absence of the internal carotid artery on the affected side.

PRIMARY SQUAMOUS CELL CARCINOMA OF THE CORNEA

REPORT OF A CASE *

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Reports of cases of epibulbar squamous cell carcinoma or epithelioma are numerous, but cases in which the tumor is primary on the cornea are sufficiently rare to warrant reporting in each instance.

There is such a confusion of terms used in the literature for this pathologic condition that it is difficult to obtain a complete bibliography. Parsons,[1] in 1904, could find only six cases reported in which the accuracy of the diagnosis of primary epithelioma of the cornea was probable. "The American Encyclopedia of Ophthalmology" [2] quotes some of these and adds nine more cases, eight being reported under epithelioma and one under carcinoma.

A review of all cases of epibulbar carcinoma involving the cornea reported since then has shown only six cases [3] in which primary involvement of the cornea could not be doubted. This brings the total number of cases reported to twenty-one. To this I wish to add the following case.

REPORT OF CASE HISTORY

History.—J. J. B., aged 74, white, a mail-carrier, was first seen on Nov. 7, 1927, complaining that his left eye had been painful and red for two or three weeks. It bothered him so much that he was unable to do his work.

About five years before, he had noticed a "small growth" starting on the inner part, which gradually crept over the eye until for two years the vision had been reduced to perception of light. The eye had been painful and red at times for two or three years when he took a "cold." During this time, several skin "growths" had been removed from his face and body by his family physician. There was no history of any previous injury or similar condition in his family.

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* Submitted for publication, March 25, 1929.

* Read before the Omaha and Council Bluffs Ophthalmological and Otolaryngological Society, Jan. 16, 1929.

1. Parsons, J. H.: The Pathology of the Eye, New York, G. P. Putnam's Sons, 1904, vol. 1, p. 263.

2. Woods, Casey: American Encyclopedia of Ophthalmology, Chicago, Cleveland Press, 1914, vol. 5, p. 33.

3. Alessandro, F.: Clin. ocul. **15**:1669, 1914. Keith, D. Y., and Keith, J. P.: Am. J. Roentgenol. **9**:337, 1922. Koyanagi: Klin. Monatsbl. f. Augenh. **67**:573, 1922. Rados, A., and Schinz, H. R.: Arch. f. Ophth. **110**:370, 1922. Finnoff, W. C.: Am. J. Ophth. **6**:505, 1923.

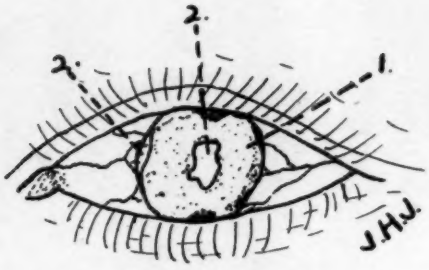

Fig. 1.—Sketch made at first examination showing: 1, ring shaped tumor; 2, uninvolved cornea.

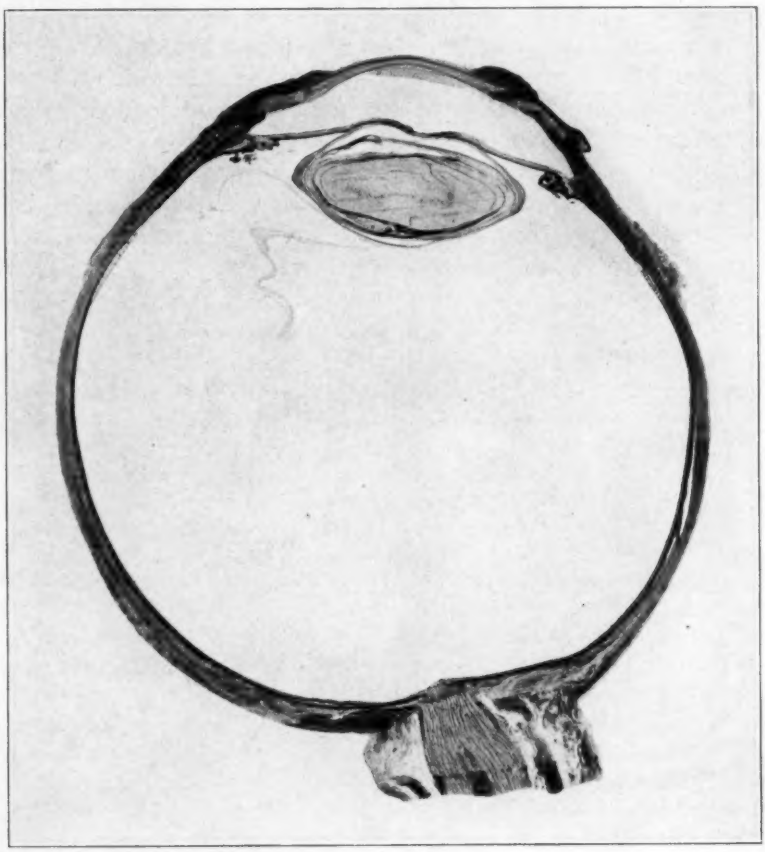

Fig. 2.—Section showing tumor limited to cornea.

Examination.—The lids of the left eye were normal. There was marked conjunctival congestion and pericorneal injection. The periphery of the cornea presented a gray, semitranslucent, irregular thickening, from 3 to 5 mm. wide, forming a complete ring about the pupillary area. At about 9:30 clear cornea was seen outside the growth, and at no point did it cross the limbus. Many vessels extended into it from the limbus. The surface of the growth was stained by fluorescein and was sharply defined from the rest of the cornea which was so cloudy as to prevent ophthalmoscopic examination. Transillumination of the globe did not reveal an intra-ocular extension of the process. The vision was limited to the ability to see movements of the hands.

In the right eye marked arcus senilis, a few peripheral radial lens opacities and marked sclerosis of the retinal vessels were noted. The vision was 20/30 with correction.

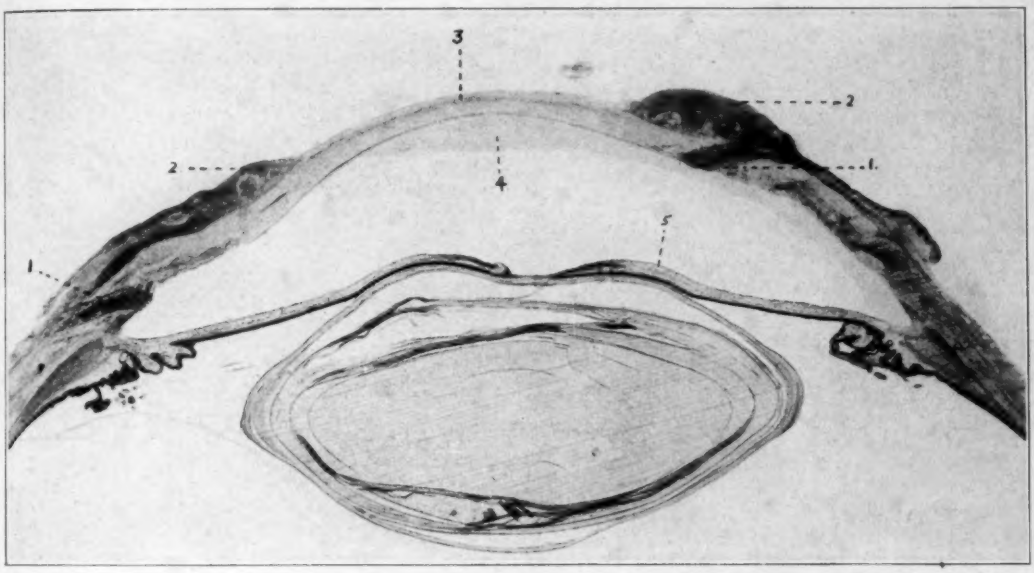

Fig. 3.—Section showing: 1, artefact from fold in section; 2, tumor; 3, leukocytic infiltration; 4, albuminous exudate; 5, atrophic iris adherent to anterior lens capsule.

The preauricular and submaxillary glands were normal. Several areas of keratosis senilis were seen on the face and body.

Enucleation was advised and accepted. This was done under local anesthesia on Nov. 10, 1927. When the nerve was severed, a hemorrhage occurred back of Tenon's capsule causing proptosis of the orbital tissue against the lids. There was practically no external bleeding. The healing was uneventful and left a large and freely movable stump.

Four weeks later, he was fitted with a prothesis and resumed his former work. Fourteen months later, there had not been any discomfort or sign of recurrence. The eye was sent to the Army Medical Museum, and I received from Major George R. Callender the photomicrographs and the following pathologic report:

Histologic Examination.—"Extending in from the limbus the corneal epithelium is somewhat thickened and shows some hydropic degeneration. Beneath this the tissues are infiltrated with lymphocytes and an occasional polymorphonuclear leukocyte. Approximately 2 mm. in from the position of the limbus on the cornea are seen small masses of squamous cell carcinoma showing a tendency to keratinization. These are prolongations of somewhat thicker masses of the same tumor cells which form a ring of thickening about the outer 3 to 5 mm. of the cornea. This carcinoma is infiltrated with lymphocytes and contains a few vessels. It represents the thickening described in the examination of the eye before removal. Masses of

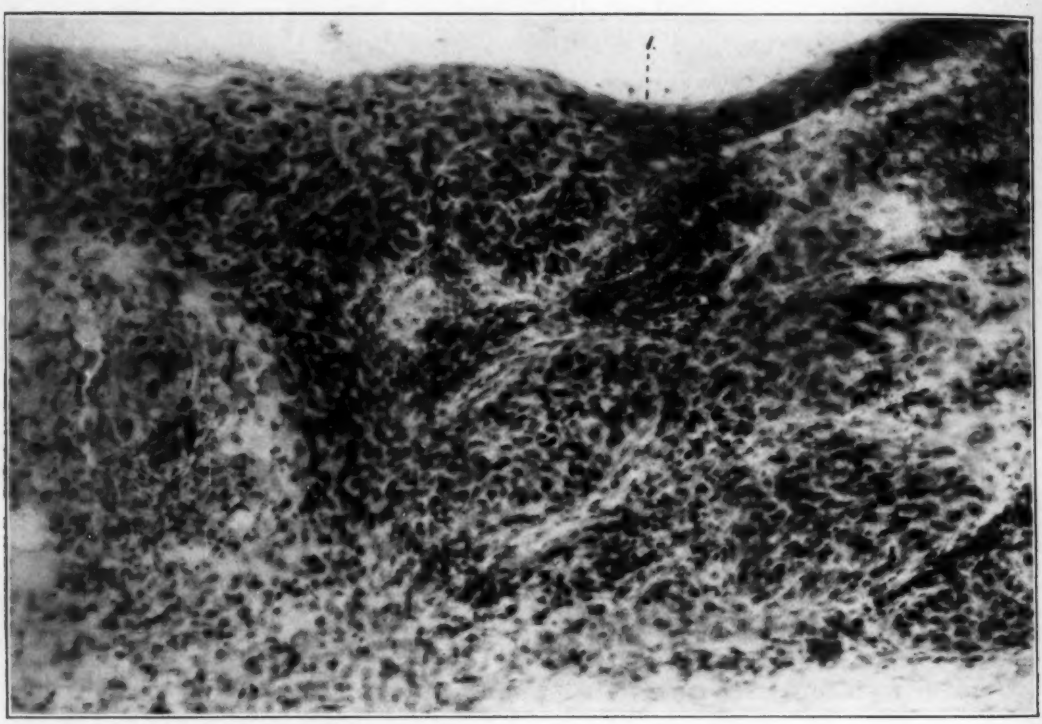

Fig. 4.—High power photomicrograph of tumor showing loss of regular arrangement of cells at 1.

tumor invade the deep lymphatics between the limbus tissue and the sclero-cornea. The area between the masses of carcinoma on the cornea is denuded of epithelium forming a shallow ulcer and the cornea beneath is infiltrated with a relatively small number of leukocytes. There is an albuminous exudate in the anterior chamber adherent to Descemet's membrane while on the latter are a few polymorphonuclear leukocytes. The iris is adherent to the capsule of the lens about the pupil. Iris is somewhat atrophic. Choroidal vessels are engorged with blood and a small amount of albuminous fluid (edema) separates the layer of rods and cones from the pigment epithelium in the macular region the tissues of which are also edematous.

"Diagnosis: Carcinoma arising in the corneal epithelium within the limbus; ulceration of the cornea with keratitis and edema of the macula. There is no evidence of extension beyond the globe."

COMMENT

According to Parsons, this type of tumor has only a slight tendency to extend deeply. Resistance is encountered at Bowman's membrane. If the substantia propria is invaded, however, it offers some resistance by setting up a barrier of embryonic connective tissue around the epithelial cells.

Extension of the growth to surrounding tissues takes place by means of the lymphatic spaces between the layers of the substantia propria to reach the lymphatics connected with the canal of Schlemm. From there it may spread to Tenon's capsule and rarely to the preauricular and submaxillary glands.

When ulceration has taken place, there is a leukocytosis due to the pyogenic bacteria on the surface.

This case is unusual in that it is the only one reported in which the tumor forms a complete ring entirely on the cornea, or in which senile keratoses of the skin were associated with the lesion of the eye.

AN OPTICAL REPLICA OF THE HUMAN EYE FOR THE STUDY OF THE RETINAL IMAGE*

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For a number of years, Prof. Adelbert Ames, Jr., of Dartmouth College, has been interested in the problem of ascertaining the nature of the retinal image in the human eye. His method of approaching this problem was to measure the aberrations in his own eyes, to study their effects as he interpreted them and so to analyze the nature of his subjective impressions.[1] For this study, I attempted to design for him a photographic lens which had the same aberrations as were present in his eye, according to his subjective measurements.[2] With a lens of this kind it was possible to approximate the chromatic aberration, spherical aberration, astigmatism and distortion of his right eye. The design, however, was for a flat focal plane and the effects were different from those found in the eye, for the retina, which is really the focal plane of the eye, is spherical in form.[3]

Furthermore, the focal length of this lens and of the other lenses that were made was much greater than that of the eye, for it seemed impracticable and unnecessary to attempt to work in the small dimensions of the eye. This difference in focal length produced object fields which did not agree with those of the eye, even though the image fields of the constructed lens were conjugate to the measured object fields of the eye. Possibly a lens could have been made with the desired object fields, but it would have been quite different in construction from the eye, and the results somewhat questionable. Moreover, if a lens of this kind were constructed it might not represent an average eye but the aberrations of an individual eye.[4]

That individual eyes differ greatly became evident at once when I attempted to repeat the experiments of Professor Ames. Although a similar color curve was obtained, I failed to get the same type of

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* Submitted for publication, March 27, 1929.

* A thesis submitted to the faculty of Dartmouth College in partial fulfilment of the requirements for the degree of Doctor of Philosophy in Physiologic Optics, June, 1926.

1. Ames, Proctor and Ames: Vision and the Technique of Art, Proc. Am. Acad. Arts & Sc., February, 1923, p. 3.

2. Ames and Proctor: Dioptrics of the Eyes, J. Optic. Soc., America, January, 1921, p. 85.

3. Salzman: Anatomy and Histology of the Human Eyeball, Chicago, Chicago Medical Book Company, p. 3.

4. Ames and Proctor (footnote 2, p. 24).

spherical aberration curve. This may have been because of insufficient measurements or because of an undilated pupil. Moreover, when I attempted to measure the positions of the primary and secondary focal lines produced by light entering the eye obliquely, I obtained unsymmetrical results.[5] This fact suggested a decentered element somewhere in the lens system, for the tipping of the fields was similar to that produced by a decentered lens in the four piece air-spaced anastigmat.[6]

This tipping of the field, with the primary and secondary focal lines in front of the retina on the temporal side, and behind on the nasal side, was found when checked one year later with improved apparatus. The effect was found also in the vertical meridian, where the field seemed to fall in front of the retina in the upper half and behind it in the lower half. Further measurements showed a tipping in my left eye similar to that in the right. This odd effect could conceivably have been produced by a tipping of the cornea or of the crystalline lens.

This gave rise to the question whether or not the best optical image was formed on the fovea.[7] Owing to the decreased sensitivity of the peripheral parts of the retina as compared with that of the macular region,[8] one might easily interpret a blurred image on the fovea as sharper than a focused image falling outside the fovea.

It is a well established fact that the optic axis of the eye passes out and down with reference to the visual axis, forming an angle alpha of from 5 to 7 degrees in the horizontal meridian, and from 2 to 3 degrees in the vertical meridian.[9] Helmholtz defined the optic axis as that line which passed through the anterior and posterior poles of the eye,[10] but Savage claimed that the optic axis started at the fovea and was coincident with the visual axis, and that there was therefore no angle alpha.[11] Tscherning developed a method for measuring the angles of the eye by his ophthalmophakometer,[12] and Dr. Lucien Howe suggested certain

5. Gliddon: Unpublished work done at Dartmouth College in 1923-1924.

6. Frederick: Unpublished work on the four piece airspace anastigmat, Hawk Eye Works, Eastman Kodak Company, Rochester, N. Y., 1912-1915.

7. Laurence: Visual Optics and Sight Testing, London, School of Optics, p. 6.

8. Parsons: An Introduction to the Study of Color Vision, New York, G. P. Putnam's Sons, p. 204.

9. Tscherning: Physiologic Optics, Philadelphia, Keystone Publishing Company, pp. 45 and 80.

10. Helmholtz: Physiological Optics, translation of third edition, Ithaca, N. Y., The Optical Society of America, vol. 1, p. 94.

11. Savage: Ophthalmic Myology, Nashville, Tenn., G. C. Savage, p. 10.

12. Tscherning (footnote 9, p. 53).

modifications in the ophthalmometer so that it might be used to approximate the angle alpha.[13]

The work done by Professor Ames and Professor Proctor at Dartmouth[14] and by the students at Rochester, N. Y., under my supervision, leaves no doubt that the average eye is a badly decentered and tipped optical system.[15] This, of course, raises the question of the definition of the optic axis of a system of this kind. Gullstrand suggested that it be defined as the normal to the cornea which passes through the center of the pupil.[16]

The effect of the angle alpha in the vertical meridian would be to produce a certain amount of astigmatism with the rule, while its effect in the horizontal meridian would introduce astigmatism against the rule.[17] This angle, therefore, would have a marked influence on the nature of the optical image. The fact that the values of the angle alpha differ slightly in individual eyes offers a plausible explanation of the discrepancy which exists between the astigmatism of an eye as measured with an ophthalmometer, and that found objectively with the skiascope or subjectively with the optometer.[18] The question whether the distortion of an eye[19] is an optical, retinal or psychologic effect has never been satisfactorily settled.[20] The nodal points of an eye have never been accurately located by measurement.[21] The questions of coma and lateral color have yet to be thoroughly investigated.[22] A thousand and one other questions might be answered if it were only possible to deal with the optical image objectively.

THEORETICAL CALCULATIONS ON THE SCHEMATIC EYE

Since the real solution to all these problems seemed to hinge on the making of an eye, and since, obviously, the human eye could not be reproduced, the question arose of how nearly one could approximate the eye with an optical replica. Furthermore, unless the replica could be justified theoretically and practically, any conclusions drawn from it might be open to question. Literature dealing with this subject is full

13. Howe, Lucien: Muscles of the Eye, New York, G. P. Putnam's Sons, p. 69.
14. Ames and Proctor: Unpublished work done at Dartmouth College, 1920-1923.
15. Gliddon: Unpublished work of eye measurements of students of the Rochester School of Optometry performed at the University of Rochester, in 1924-1925.
16. Helmholtz (footnote 10, p. 315).
17. Laurence (footnote 7, p. 142).
18. Tscherning (footnote 9, p. 150).
19. Tscherning (footnote 9, p. 260).
20. Tscherning (footnote 9, p. 264).
21. Helmholtz (footnote 10, p. 114).
22. Ames and Proctor (footnote 2, p. 24).

of schematic eyes and reduced eyes, but how near the truth are they? Helmholtz gave the values of Listing's schematic eye [23] which Gullstrand modified by inserting a core in the lens.[24] Hans Gertz gave slightly different values for the core and the lens, based on simpler mathematical theory.[25] Tscherning gave values for the eye [26] which were accepted by French [27] and by Sheard.[28] These did not include a core, since the question of the change in shape of the lens during the act of accommodation seemed to involve the core. Without it, the power of the dynamic eye and the equivalent index of the lens could not be satisfactorily reconciled to one another.[29]

Descartes seems to have been the first to understand the structure of the eye and the optical properties of its parts.[30] Since that time, many investigators have attacked the problem of determining the constants of the eye; that is, the radii of curvature of the cornea and crystalline lens, the indexes of the lenses and humors and their thickness. Of the values given by these investigators, those of Gullstrand and Tscherning are probably nearest the truth.

For this work it seemed best to use the aberrations of the eye as a criterion. First, the schematic eyes of Gullstrand and Tscherning were triangulated to see if the aberrations were materially influenced by the core. Table 1 gives the constants of the two eyes and the aberrations resulting along a ray aimed at the center of the pupil in each case. Figure 1 represents the two schematic eyes drawn three times their actual size.

The distortion and position of the fields are computed with reference to a plane perpendicular to the optic axis at F_2, and do not represent the true values in an eye. The latter are obtained when the retina is drawn in its probable position. The difference between the values in the two columns of table 1 is so slight, however, that for the static eye no error is introduced in taking the data of Tscherning.

The schematic eye of Tscherning was then triangulated completely and showed the curves for spherical aberration and sine condition that appear in figure 2. The values are also given in table 2. These are the characteristic curves of an uncorrected lens. Since the spherical aberration and sine condition are of the same general shape one would expect relatively little coma in the image.[6]

23. Helmholtz (footnote 10, p. 95).

24. Von Rohr: Eyes and Spectacles, London, Hatton Press, p. 3.

25. Gertz, Hans: Déduction élémentaire du système optique équivalent du cristallin, Acta ophth. **2**:137, 1924.

26. Tscherning (footnote 9, p. 33).

27. French: The Unaided Eye, Tr. Optical Soc., London, April, 1919, p. 212.

28. Sheard: Physiologic Optics, Chicago, Cleveland Press, p. 37.

29. Von Rohr (footnote 24, p. 4).

30. Descartes: Opera Philosophica, 1656, chap. 3.

The complete triangulation for the fields gave the results in table 3; these are shown graphically in figure 3.

It is evident at once that the fields of the eye straddle the retina, if the latter is taken to be spherical with a radius equal to one-half the diameter of the eyeball. This is the value usually accepted [3] and gives:

$$R = \tfrac{1}{2}(\underset{t_1}{1.15} + \underset{t_2}{2.39} + \underset{t_3}{4.06} + \underset{t_4}{17.15}) = 24.75/2 = 12.38 \text{ mm.}$$

This position of the field is in agreement with the work of different experimenters [31] and brings the best image on the retina even out to 20 degrees.

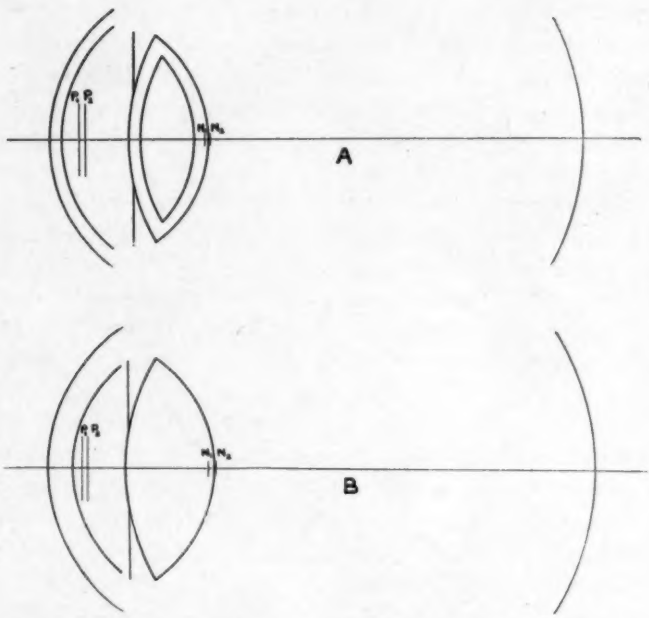

Fig. 1.—*A*, eye of Gullstrand; *B*, eye of Tscherning. N_1 is the first nodal point, N_2 the second nodal point, P_1 the first principal point, and P_2 the second principal point.

In order to compare this value with those of Matthiessen,[32] a chief ray was triangulated. This was a ray which actually passed through the center of the pupil. It gave the following results for Tscherning's schematic eye. (*a*) A ray aimed at center of pupil ($u_1 = -3.54$)[33]: distortion 20 degrees, —0.0893; primary focal line, —1.6398, and

31. Ames and Proctor (footnote 2, p. 67).

32. Matthiessen: Ueber der geometrische Gestalt der theoretischen Retina des periskopischen schematischen Auges, Arch. f. Ophth. **25**:257, 1879.

33. U_1 is the object distance measured from the first surface.

TABLE 1.—*Constants and Aberrations of the Schematic Eyes of Gullstrand and Tscherning**

Eye of Gullstrand				Eye of Tscherning			
	Mm.				Mm.		
R_1	— 7.70			R_1	— 7.98		
R_2	— 6.80			R_2	— 6.22		
R_3	—10.00			R_3	—10.20		
R_4	— 7.91						
R_5	+ 5.76						
R_6	+ 6.00			R_4	+ 6.17		
t_1	+ 0.50			t_1	+ 1.15		
t_2	+ 3.10			t_2	+ 2.39		
t_3	+ 0.55						
t_4	+ 2.42			t_3	+ 4.06		
t_5	+ 0.64						
t_6	+17.18			t_4	+17.15		
N_1	1.000	N_2	1.376	N_1	1.000	N_2	1.377
N_3	1.336	N_4	1.386	N_3	1.3365	N_4	1.420
N_5	1.406	N_6	1.386				
N_7	1.336			N_5	1.3365		
F_1	+17.06			F_1	+17.13		
F_2	—22.78			F_2	—22.89		
P_1	— 1.35			P_1	— 1.54		
P_2	— 1.60			P_2	— 1.86		
N_1	— 7.08			N_1	— 7.30		
N_2	— 7.33			N_2	— 7.62		
S.A. F/3.8	— 1.3787			S.A. F/3.8	— 1.2895		
S.C. F/3.8	— 1.6392			S.C. F/3.8	— 1.5180		
Coma F/3.8	— 0.2605			Coma F/3.8	— 0.2285		
S.A. F/5.7	— 0.5807			S.A. F/5.7	— 0.5433		
S.C F/5.7	— 0.6971			S.C. F/5.7	— 0.6448		
Coma F/5.7	— 0.1164			Coma F/5.7	— 0.1015		
Dist. 20°	— 0.0545			Dist. 20°	— 0.0893		
20° p	— 1.6585			20° p	— 1.6398		
20° s	— 1.2202			20° s	— 1.2119		

* R represents the radius; t, the thickness; N, the relative index; F, focal distance; F_1, the anterior focal length; F_2, the posterior focal length; P_1, the first principal point; P_2, the second principal point; N_1, the first nodal point; N_2, the second nodal point; S.A., the spherical aberration; S.C., the sine condition; Dist., the distortion; 20°, the field at 20 degrees; p, the primary focal line, and s, the secondary focal line.

TABLE 2.—*Sine Condition and Spherical Aberration of the Eye of Tscherning*

Speed	Sine Condition	Spherical Aberration	Coma
F/ 3.8	—1.5180	—1.2895	—0.2285
F/ 4.5	—1.0567	—0.8937	—0.1630
F/ 5.7	—0.6448	—0.5433	—0.1015
F/ 6.8	—0.4544	—0.3835	—0.0709
F/ 8.0	—0.3217	—0.2704	—0.0513
F/11.0	—0.1670	—0.1409	—0.0261
F/16.0	—0.0785	—0.0661	—0.0124
F/22.0	—0.0403	—0.0344	—0.0059

TABLE 3.—*Results of the Complete Triangulation for the Fields of the Eye of Tscherning*

Field, Degrees	Distortion	Primary Focal Line	Secondary Focal Line
5	—0.0012	—0.1056	—0.0768
10	—0.0106	—0.4224	—0.3081
15	—0.0375	—0.9308	—0.6805
20	—0.0893	—1.6398	—1.2119

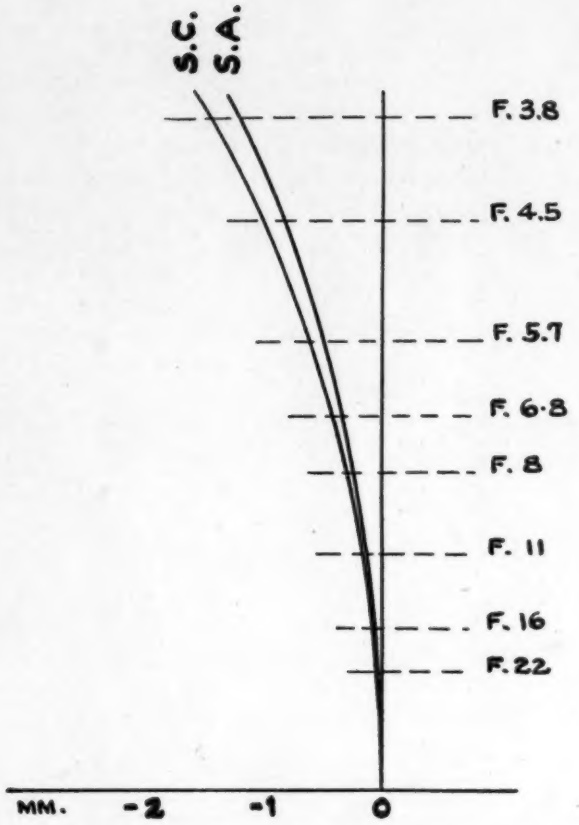

Fig. 2.—Curves for sine condition and spherical aberration of the eye of Tscherning. S.C. is the sine condition; S.A., the spherical aberration, and F, the focal distance.

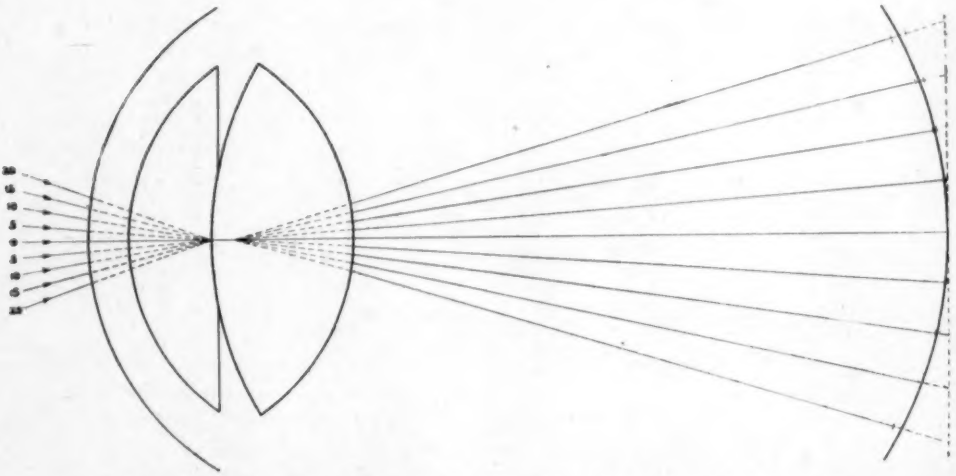

Fig. 3.—Eye of Tscherning. Results in complete triangulation of the fields of the eye.

secondary focal line, —1.2119; and (*b*) a chief ray through the center of the pupil ($u_1 = {}^{-}2.94$): distortion 20 degrees, —0.1065; primary focal line, —1.7250 and secondary focal line, —1.2397. This shows that the difference in distortion is negligible as is the change in the field, and that although the image is formed along the chief ray of a bundle, focal length and the different aberrations are measured with reference to the nodal points. The schematic eye was triangulated, therefore, by using rays aimed at the first nodal point. The results shown in table 4 were obtained ($u_1 = {}^{-}7.30$). These values were computed with reference to a flat plane as before. The position of the fields relative to the retina is shown in figure 4.

This shows that for practical purposes of comparison any set of rays may be taken and that the fields computed along a ray aimed at

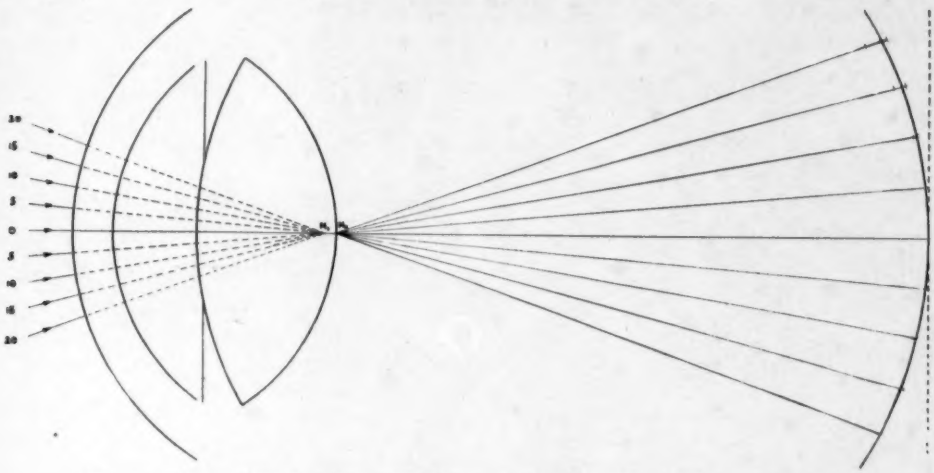

Fig. 4.—Eye of Tscherning. Position of the fields in relation to the retina when the eye is triangulated with rays aimed at the first nodal point; × 6.

the center of the pupil are not materially different from fields along a ray aimed at the nodal points. As a matter of fact, the center of gravity of the bundle of light passing through the pupil would probably be the ray which passes through the center of the pupil.[6] Gullstrand claimed, however, that the optical center of the pupil was above the anatomic center; in that case, the chief ray would pass through the upper part of the pupil.[34]

Gullstrand and Tscherning both claimed that the eye focused that part of the cone of light in which the section of the caustic was smallest.[35] Ames and Chapman, however, both found that their eyes focused that

34. Helmholtz (footnote 10, p. 431).

35. Tscherning (footnote 9, p. 129).

part of the cone which had the smallest diameter.[36] For the present purposes, however, these are fine points of difference, and this study is concerned rather with the nature of the retinal image in its entirety, as formed by the spherical surfaces used. It is not justifiable, therefore, to try to generalize from the results with one or two rays. The data thus obtained are to be used merely for comparative purposes in building a replica which will approximate the schematic eye of Tscherning.

These aberrations were worked out for the *D* line, and so are monochromatic. When the dispersion of the eye was considered, it was found that the humors as well as the lens and cornea had approximately the same power of dispersion as water. If a value of V = 56.6 [37]

TABLE 4.—*Results of Triangulation of the Schematic Eye Using Rays Aimed at the First Nodal Point*

Field, Degrees	Distortion	Primary Focal Line	Secondary Focal Line
5	+0.0003	—0.1190	—0.0814
10	+0.0028	—0.4767	—0.3266
15	+0.0067	—1.0676	—0.7304
20	+0.0166	—1.8812	—1.2832

TABLE 5.—*Indexes Used for the Cornea, Humors and Lens* *

Cornea	Humors	Lens
N_C 1.3750	N_C 1.3346	N_C 1.4178
N_D 1.3770	N_D 1.3365	N_D 1.4200
N_F 1.3816	N_F 1.3406	N_F 1.4250

* In this table and the accompanying tabulations, N_C indicates the index of refraction for C light; N_D, the index of refraction for D light; N_F, the index of refraction for F light; V, the aspercivity or $\frac{N_D - 1}{N_F - N_C}$; F_C, the focal length for C light; F_D, the focal length for D light; F_F, the focal length for F light; BF_C, the back focus for C light; BF_D, the back focus for D light, and BF_F, the back focus for F light.

is used for the water, one can compute at once the chromatic aberration of the schematic eye. This is equivalent to using the indexes shown in table 5 for the different parts of the eye. This means that the eye can be considered as built of substances with the following optical properties:

Cornea	N_D	1.377	V	57.12
Humors	N_D	1.3365	V	57.25[38]
Lens	N_D	1.4200	V	58.33

36. Ames and Proctor (footnote 2, p. 47).

37. Helmholtz (footnote 10, p. 173). Laurence (footnote 7, p. 285).

38. Gleichen: The Theory of Modern Optical Instruments, London, His Majesty's Stat. Off., p. 7.

With these values the following focal lengths were obtained for Tscherning's schematic eye:

F_C	22.9664		BF_C	17.2314	
		0.0780			0.0815
F_D	22.8884		BF_D	17.1499	
		0.1831			0.1863
F_F	22.7053		BF_F	16.9636	

This gives a color curve which is in excellent agreement with curves obtained empirically by different experimenters.[39]

CONSTRUCTION OF THE REPLICA

As soon as theory showed that the data of Tscherning could be used for the schematic eye, and that the aberrations of an eye of this kind could be studied, the problem which first arose was that of finding the substances to be used for the cornea (N_D 1.377) and the crystalline lens (N_D 1.42). The ordinary optical tables [40] failed to show any substances with the desired properties, so the possibility of raising all

TABLE 6.—*Values of Substances to Be Used for the Cornea and Crystalline Lens of the Replica*

Values Sought		Values, with Water for the First Medium		Values with Special Glasses	
N_1	1.0000	N_1	1.3365	N_1	1.1438
N_2	1.3770	N_2	1.8400	N_2	1.5750
N_3	1.3365	N_3	1.7862	N_3	1.5287
N_4	1.4200	N_4	1.8978	N_4	1.6242
N_5	1.3365	N_5	1.7862	N_5	1.5287

indexes and still keeping their relative values was next considered. The desired values are given in the first column of table 6.

If water were used instead of air for the first medium, one would obtain the values in the second column of table 6. It is apparent at once that this would require special glasses for the cornea and the crystalline lens; and, while they might be made, they would be flint glasses and so would have too great a dispersive value. Furthermore, it would be hard to find clear liquids of proper transparency for the humors; they also would have too high a dispersion.

The next possibility was to use light barium crown glass for the cornea, and dense flint glass for the crystalline lens. This would mean using a liquid of index 1.1438 in place of air. Chemists reported, however, that no such liquid existed. The necessary values are given in the third column of table 6.

39. Gliddon (footnote 5). Hartridge, H.: Chromatic Aberration and the Resolving Power of the Eye, J. Physiol. **52**:175, 1918.

40. Landolt-Börnstein: Physikalisch-chemische Tabellen, Berlin, Julius Springer.

It was necessary, then, to return to the original values given in column 1. An exhaustive search was made for substances of index 1.377 and index 1.42. Although every possible source was tried, a substance of this kind could not be found, and liquids had to be used. This meant that the cornea had to be made of two thin shells, the space between filled with a liquid of index 1.377, and the crystalline lens made in a similar manner. Experiments were performed with numerous transparent substances for the shells but it was found that glass was the only practical material. Helmholtz proved that if a layer of a

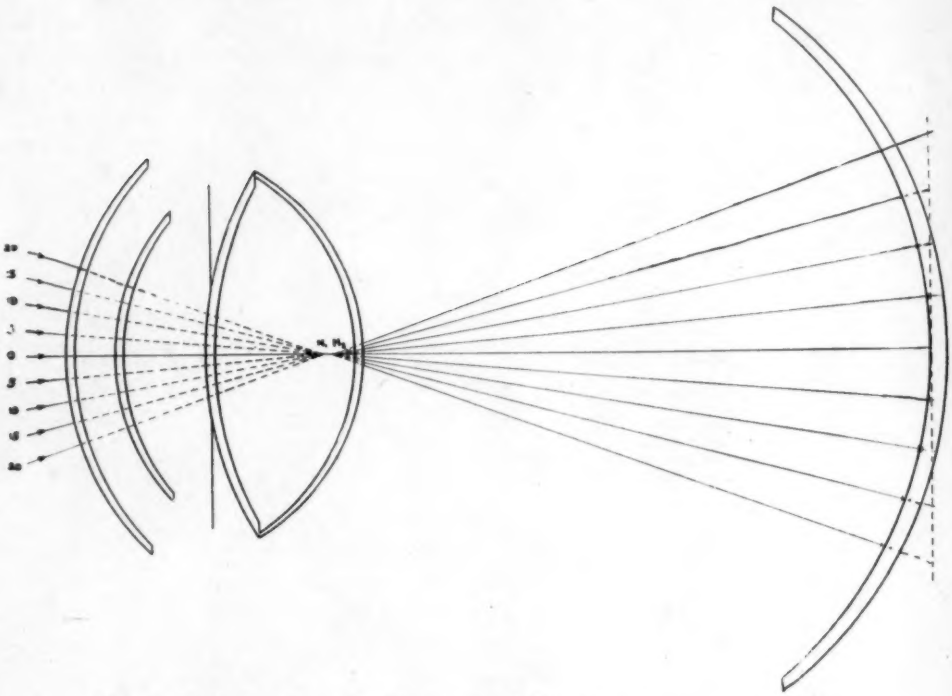

Fig. 5.—Optical replica. Position of the fields with reference to the retina.

substance was thin enough its optical effect was practically nil.[41] The next object was to find how thin it was possible to make the glass shells. The limit seemed to be 0.25 mm., so the theory was computed on this basis, concentric surfaces being used for each shell first, and then surfaces with the same radii. The latter produced aberrations near enough to those of the triangulated eye so that no appreciable error was introduced by the thickness of the shells. It also seemed slightly better to increase the over-all dimensions of the eye by the thickness of the shells rather than to decrease the thickness of the humors. The first column in

41. Helmholtz (footnote 10, p. 85).

table 7 gives the data for the eye as previously determined, while the second column gives the data for the proposed replica. These values were obtained by the triangulation of rays aimed at the first nodal point. Figure 5 shows the position of the fields with reference to the retina for the replica.

TABLE 7.—*Comparison of Data on the Eye and Replica* *

Eye		Replica	
F_A	+17.13	F_A	+17.23
F_P	−22.89	F_P	−23.02
FF	−15.59	FF	−15.60
BF	−17.15	BF	−16.35
P_1	− 1.54	P_1	− 1.63
P_2	− 1.86	P_2	− 1.93
N_1	− 7.39	N_1	− 7.42
N_2	− 7.62	N_2	− 7.72
S.A. F/5.7	− 0.5433	S.A. F/5.7	− 0.5178
S.A. F/6.3	− 0.4474	S.A. F/6.3	− 0.4189
S.A. F/8.0	− 0.2704	S.A. F/8.0	− 0.2543
S.C. F/5.7	− 0.6448	S.C. F/5.7	− 0.6339
S.C. F/6.3	− 0.5312	S.C. F/6.3	− 0.5129
S.C. F/8.0	− 0.3217	S.C. F/8.0	− 0.3113
Coma F/5.7	− 0.1015	Coma F/5.7	− 0.1161
Coma F/6.3	− 0.0838	Coma F/6.3	− 0.0940
Coma F/8.0	− 0.0513	Coma F/8.0	− 0.0570
Dist. 5°	+ 0.0003	Dist. 5°	+ 0.0003
Dist. 10°	+ 0.0028	Dist. 10°	+ 0.0004
Dist. 15°	+ 0.0067	Dist. 15°	+ 0.0013
Dist. 20°	+ 0.0168	Dist. 20°	+ 0.0027
5°	− 0.1190 − 0.0814	5°	− 0.0975 − 0.0743
10°	− 0.4767 − 0.3266	10°	− 0.3924 − 0.2996
15°	− 1.0676 − 0.7304	15°	− 0.8781 − 0.6704
20°	− 1.8812 − 1.2832	20°	− 1.5348 − 1.2165

* F_A indicates the anterior focal length; F_P, the posterior focal length; FF, the front focus, and BF, the back focus. At the bottom of the table, the first figure in each bracketed group indicates the primary focal line; the second, the secondary focal line.

TABLE 8.—*Fields Formed by a Chief Ray Through Center of Pupil of Replica and Tscherning's Schematic Eye*

Eye	Object Distance	Field, Degrees	Distortion	Primary Focal Line	Secondary Focal Line
Schematic	−2.9400	20	−0.1065	−1.7250	−1.2397
Replica	−3.4220	20	−0.0942	−1.5612	−1.1899

Lastly, it seemed best to compare the field formed by a chief ray which passed through the center of the pupil of the replica with the field formed by a chief ray passing through the schematic eye of Tscherning. Triangulation gave the results shown in table 8. This

shows that within the limits of measurement, the theory of the replica is in satisfactory agreement with the theory of Tscherning's schematic eye.

The liquids used for the eye were water and glycerin, with the optical properties shown in table 9.[42]

TABLE 9.—*Optical Properties of Water and Glycerin*

Water		Glycerin	
N_C	1.3317	N_C	1.4688
N_D	1.3336	N_D	1.4711
N_F	1.3378	N_F	1.4766
V	54.7	V	60.3

When these liquids were used in the theory of the replica they gave the following results:

F_C	23.1005	BF_C	16.4301
	0.0782		0.0822
F_D	23.0223	BF_D	16.3479
	0.1837		0.1873
F_F	22.8386	BF_F	16.1606

These values gave the same color curve as was found in Tscherning's schematic eye.

F_C	22.9664	BF_C	17.2314
	0.0780		0.0815
F_D	22.8884	BF_D	17.1499
	0.1831		0.1863
F_F	22.7053	BF_F	16.9636

The lenses were made by the Goerz Optical Company of New York, and were mounted and assembled by Mr. Ernest Robes of the Scientific Department of Dartmouth College. The replica consists of a copper cell (*A*) (figs. 6 and 7), open at the top, with a hole in the back surface (*B*) into which screws a mount holding a glass retina 0.5 mm. thick, with a radius of curvature of 12.5 mm. (*C*). This mount can be moved in and out so that the images can be studied in axial ametropia.[43] The front surface of the box contains a hole (*D*) in which may be placed different sized diaphragms (*E*). In front of this is an iron holder (*F*) which encloses another iron holder (*G*) with the cell containing the cornea (*H*). These holders of iron are magnetized and are so constructed that as one slips inside the other, the cornea may be tipped through any angle desired and will stay in that position. A similar arrangement was made for the crystalline lens and both holders were mounted so that either cornea or lens, or both, can be tipped from 0 to 10 degrees and decentered, with reference to the optic axis of

42. Thompson: Optical Tables and Data, New York, Spon and Chamberlain, p. 27.

43. Sheard: American Encyclopedia of Ophthalmology, Chicago, Cleveland Press, vol. 13, p. 9806.

the system, up to 5 mm. A small hole was bored through the top of the mount of the lens to represent the canal of Schlemn, so that when the posterior part of the eye was filled with liquid some of this liquid would filter into the anterior chamber and so completely fill the artificial eye. Four windows were placed in the back (*I*) of the replica so that it would be possible to see the interior and make whatever adjustments were desired.

There remained the problem of filling the cornea and the crystalline lens. The lenses of the cornea were mounted with the proper separation; the space between was filled, under a vacuum pump, through a hole bored in the mount. In the case of the crystalline lens this could not be done, for the two shells were mounted with their edges in contact. The only way of filling the lens was through holes in one of the surfaces. There was then the problem of drilling holes in pieces

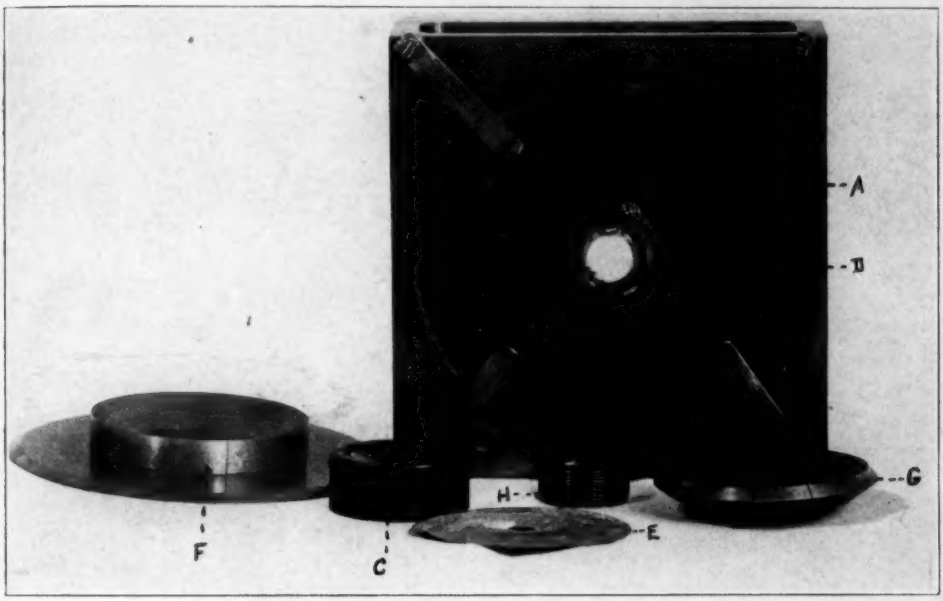

Fig. 6.—Front view of replica with parts unassembled. *A* indicates the copper cell; *C*, the retina; *D*, the opening for insertion of the diaphragms; *E*, the diaphragms; *F*, the outer iron holder; *G*, the inner iron holder, and *H*, the cell containing the cornea. Slight reduction in this and subsequent figures.

of glass as thin as these. After two attempts, the foreman of the Goerz plant ground the shells out of a piece of glass which contained two small air bubbles the desired distance apart. After the shells were finished, he ground out the air bubbles, thus producing the desired holes. The shells were then cemented together and the lens was filled under the vacuum pump. The holes were sealed with beeswax.

The liquids used were glycerin and water mixed in different proportions to give the desired indexes, the latter being checked with a Zeiss Abbe refractrometer for a period of three days, and always measured at room temperature

(22 C.).[44] A small amount of each liquid was sealed for three months; it was then tested and found to vary less than three points in the fourth decimal place, which is about the limit of accuracy of the instrument and far more than the accuracy necessary for the experiment.

The accompanying photographs show the construction of the replica and the way in which the different units were mounted. Figures 6 and 7 represent front and rear views with parts unassembled; figures 8 and 9 are front and rear views with parts assembled; figure 10 is a side view, and figure 11 a top view. All pictures show approximately the actual size of the replica and indicate the bulky structure necessary to mount these small lenses and to have the necessary adjustments for tipping and decentering. The calibrations of the instrument, both for tipping and for decentering, are seen in figure 8.

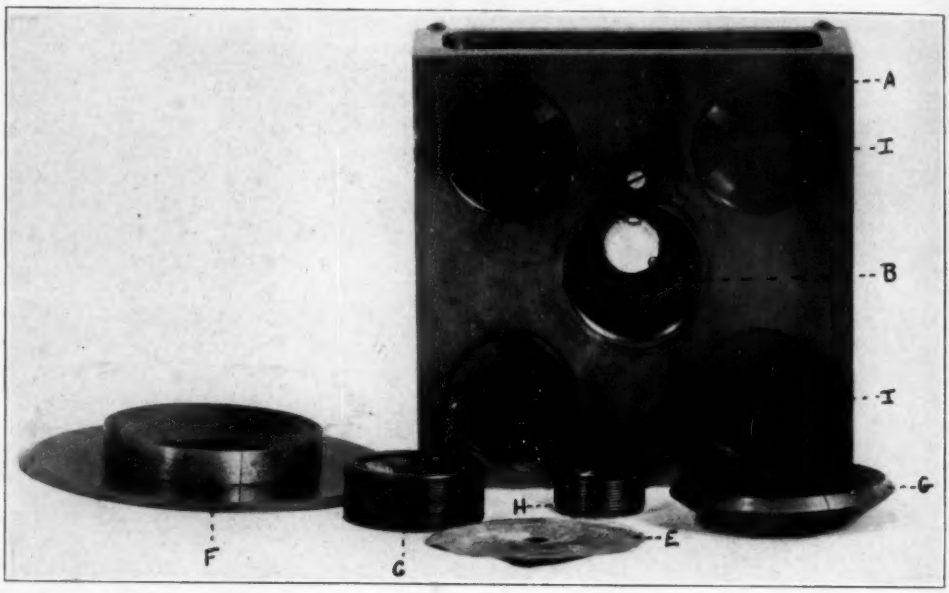

Fig. 7.—Rear view of replica with parts unassembled. In addition to the parts identified in figure 7, there are: *B*, the opening for insertion of the mount holding the retina, and *I*, the windows.

MEASUREMENTS ON THE REPLICA

The eye was assembled according to theory with a 4 mm. pupil (its average diameter) [45] and examined on the lens bench at Dartmouth in September, 1925. When it was rotated around the second nodal point (as computed by theory) the image seemed to fall on the glass retina out to 20 degrees, but this reading was questionable because the edge

44. Houstoun: A Treatise on Light, New York, Longmans, Green & Company, p. 107.

45. Tscherning (footnote 9, p. 42).

Fig. 8.—Front view of replica with parts assembled.

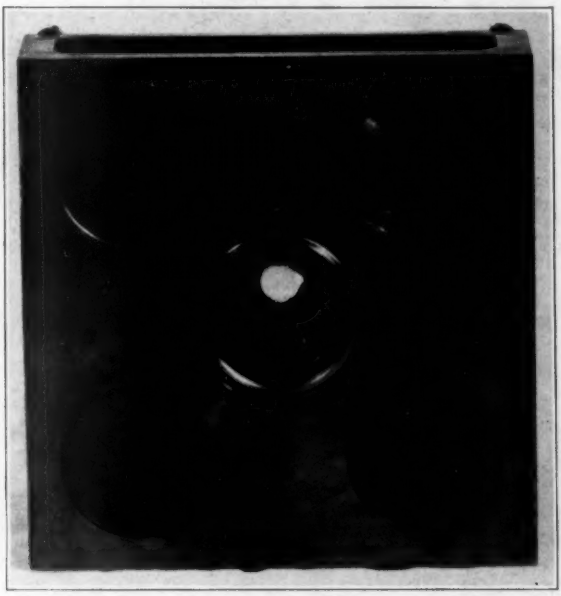

Fig. 9.—Rear view of replica with parts assembled.

of the mount obstructed the pencil of light. Accordingly, the mount was cut down 5 mm., and the lens was assembled in Rochester, N. Y., in January, 1926. Measurements of the oblique aberrations were made on the lens bench [46] at the Hawk Eye Works of the Eastman Kodak

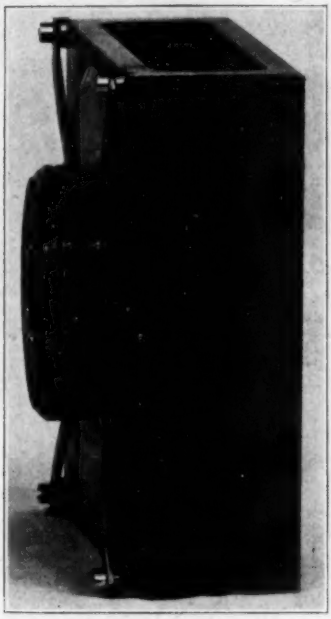

Fig. 10.—Replica from the side.

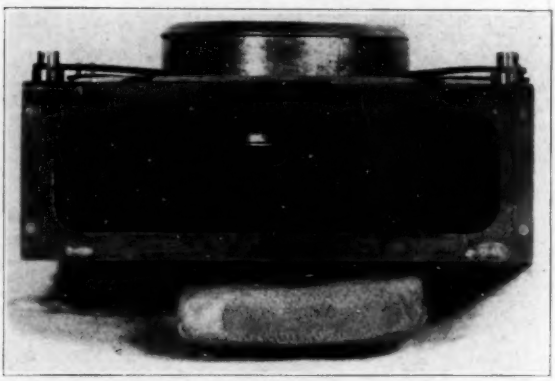

Fig. 11.—Top of replica.

Company, with an Altman objective F/3.5, 1 inch (2.5 cm.) focus, and an eyepiece that gave a magnification of 100. For the measurement of spherical aberration and chromatic aberration, an 8 mm. objective

46. Eastman Kodak Company publication concerning lenses.

was used with the same eyepiece, giving a magnification of 300. The object was a point source of light 0.75 mm. in diameter and 75 feet (214.9 meters) away.

The measurements of axial aberrations with high power objective (zero reading of the microscope, 35.1 mm.) are given in table 10.

TABLE 10.—*Axial Aberrations with High Power Objective*

	Bench Readings	Corrected Readings	
F of red	53.10	18.00	Size of haze 0.08 mm.
F of D	52.75	17.65	Size of center 0.005 mm.
F of green	52.60	17.50	
BF	53.10	18.00	

TABLE 11.—*Axial Aberrations with the Altman Objective* *

	Bench Readings	Corrected Readings
F of red	56.8	18.2
F of D	56.1	17.5
F of green	56.1	17.5

* The haze, 0.08 mm. in size, disappeared when the microscope was moved to 55.7 mm.; 56.1 minus 55.7 equals 0.4 mm. of spherical aberration.

TABLE 12.—*Bench Readings of the Oblique Fields for D Light*

Field, Degrees	Focal Length*	Back Focus	Best Image	Size of Image
0	56.10	56.40	56.10	0.036
+ 5	56.10	56.20	55.80	0.036
+10	56.05	56.05	55.80	0.027
+15	55.86 55.92	56.00	55.80	0.024
+20	55.60 55.90	55.85	55.75	0.018
— 5	56.10	56.15	55.80	0.030
—10	56.00	56.10	55.80	0.024
—15	55.68 55.90	55.90	55.70	0.018
—20	55.35 55.75	55.70	55.60	0.018

* The first reading in each bracketed group indicates the primary focal line; the second, the secondary focal line.

The haze was caused by the spherical aberration. It disappeared as the microscope was moved to 52.5; 52.8 minus 52.5 equals 0.3 mm. of negative spherical aberration, which is in agreement with theory. The true focal length was obtained by subtracting the zero reading of the microscope from the bench reading.

With the Altman objective (zero of microscope, 38.6 mm.), the measurements given in table 11 were obtained.

Table 12 shows the bench readings of the oblique fields for *D* light.

The replica was taken apart and reassembled several times at intervals of two or three weeks. In each case, however, the focal length came out the same and the images had the same characteristics as the foregoing. It seems justifiable, therefore, to take them as average characteristics of the replica. The average field readings, reduced, appear in table 13.

Focal Length.—The focal length for the D line measured 17.5 mm. when viewed through the glass retina. This, however, had a slight optical effect, and increased the optical path by about one-third the thickness of the glass: $\frac{0.5}{3} = 0.17$ mm. This would give $17.5 - 0.17 = 17.23$ mm.; this is in excellent agreement with theory, which demands 17.23. This is the reduced posterior focal length[47] for $\frac{23.02}{1.3365} = 17.23$ mm. It is necessary to compute reduced distances when working with the eye, for light entering the eye emerges finally into the vitreous,

TABLE 13.—*The Average Field Readings Reduced*

Field, Degrees	Focal Length*	Back Focus	Best Image	Size of Image
0†	17.50	17.80	17.50	0.036
5	17.50	17.58	17.20	0.033
10	17.42	17.47	17.20	0.026
15	17.17 17.31	17.35	17.15	0.021
20	16.87 17.22	17.18	17.08	0.018

* The first reading in each bracketed group indicates the primary focal line; the second, the secondary focal line.
† Distortion at 20 degrees equals 0.01 mm. barrel.

while light leaving the eye emerges into the air. The eye and the oil immersion microscope are the two big problems in the optics of nonhomogeneous mediums. The back focus measured 17.8 while the theory demanded 17.73 mm.

The nature of the image formed is as follows:

Size of the Axial Image.—The haze due to spherical aberration being disregarded, the central image was found to consist of a round disk with a diameter of 0.005 mm., and the first Newton ring with a diameter of 0.015 mm. From diffraction, one would expect the first dark ring to have a radius of 0.003 mm.[48] or a diameter of 0.006 mm. This is also in agreement with the resolving power of the eye itself which is usually taken as 1 minute of the arc.[49]

Chromatic Aberration.—The chromatic aberration curves came out in agreement with theory and with measurements made by numerous

47. Helmholtz (footnote 10, p. 277).
48. Houstoun (footnote 44, p. 84).
49. Tscherning (footnote 9, p. 334).

observers.[50] This was expected, as the same liquids were used in the replica as in the theory.

Spherical Aberration.—With a pupil measuring 4 mm. the speed of the lens was F/5.7. (Actually the speed would be F/4.3, for the reduced focal length must be used. Since the first mentioned speed notation was used in the previous part of the present work, it is continued.) Theory showed for this ray a spherical aberration of —0.5178/1.3365 = —0.3874, while measurements with a high power objective showed it to be —0.3 and with a low power objective, —0.4. An aberration of this size would ordinarily form so large a circle of confusion that all critical definition would be spoiled. If this aberration were present in the eye, one could not see even ordinarily well. The concentration of light in the image, however, is all in the central zone, with little in the circles of spherical aberration diffusion. In all probability, therefore, the eye would ignore the blurring effect and would interpret only the sharp center. As a matter of fact, in the eye itself,

TABLE 14.—*The Distance of the Best Image from the Retina*

Field, Degrees	Distance of Best Image, Mm.	Distance of Retina	Distance of Best Image from Retina
5	17.33	17.30	+00.03
10	17.03	17.08	—00.05
15	17.03	16.97	+00.06
20	16.91	16.68	+00.23

this spherical aberration is materially reduced, probably to almost nothing, by the immediate flattening of the cornea outside the optical area.[51]

Coma.—The oblique images of the replica were remarkably free from coma. In all probability one could justifiably conclude that there is relatively little coma in the eye. One would expect this from theory, but the problem warrants a much more thorough investigation.

Lateral Color.—There was no measurable amount of lateral color in the replica. It is probably present in the eye to a slight extent but is not readily visible because of the structure of the retina and its different sensitivity in different parts.[52]

Curvature of the Field.—The best image fell on the retina out to 20 degrees as theory indicated it should, for the measurements given in table 14 were obtained on the replica. There is a tendency for the best image to fall behind the retina beyond 20 degrees. This is due to the fact that the lens is rotated about the second nodal point, which is 17.23 mm. from the retina, while the center of curvature of the

50. Ames and Proctor (footnote 2, p. 51).

51. Helmholtz (footnote 10, p. 314).

52. Helmholtz (footnote 10, p. 187).

retina was 12.5 mm. away. This tendency was expected and is shown in figure 5.

Oblique Astigmatism.—The primary and secondary focal lines fell in front of and behind the retina, respectively, but no astigmatism was measurable until 15 degrees was reached. At this point the measurements given in table 15 were obtained. The best image was approximately half way between at all times. Both the primary and the secondary images were clean and straight; they were nearly free from coma, and were characteristic of a well designed lens.

Distortion.—There was no optical distortion in the replica, for the image remained in the center of the fields as the lens was rotated around the second nodal point. At 20 degrees a trace of barrel distortion developed, but this would never be of sufficient magnitude to be noticed by the eye.

TABLE 15.—*Measurements of Astigmatism in the Replica*

Field, Degrees	Theoretical Measurements*		Measurements	
15	16.92 17.13	0.21	17.00 17.14	0.14
20	16.68 17.02	0.34	16.70 17.05	0.35

* The first reading in each bracketed group indicates the primary focal line; the reading below it, the secondary focal line.

The measurements show the replica to be in sufficient agreement with theory so that the theory may be used as though it were fact. From the point of view of lens design, the eye is remarkable. This is in accord with Gullstrand's explanation of Helmholtz's famous dictum that the monochromatic aberrations of the eye are such as would not be tolerated in any good optical instrument.[53]

PROBLEMS SOLVED BY THE REPLICA

The present work shows that it is possible to construct an optical replica of the human eye. In this replica the same indexes were used as are present in the eye, but the curves of the different surfaces were spherical and to that extent different from those of the eye. The statement is justifiable, however, that the replica represents the eye if only the optical area of the cornea is considered. It seems, then, that the following statements have been verified by this work:

1. It is possible to construct an optical replica of the human eye, using the spherical curves and proper indexes of the eye.

53. Helmholtz (footnote 10, p. 440).

2. This replica has optical constants which check the theoretical calculations within the limits of measurement.

3. Tscherning's treatment of the crystalline lens with an equivalent index of 1.42 is optically correct for the static eye.

4. For the treatment of the static eye, it is unnecessary to assume a core in the crystalline lens as Gullstrand did.

5. The spherical aberration of the human eye is a negligible factor.

6. The lateral chromatism of the eye is negligible.

7. There is probably little or no coma in the human eye.

8. There is no appreciable optical distortion in the eye.

9. The best image formed along oblique rays of light entering the eye falls on the retina out to 20 degrees.

10. Diffraction is the ultimate factor of control in the formation of the optical image on the retina.

11. The aberrations of the eye do not materially reduce its resolving power.

12. The resolving power of the eye, considered as an optical instrument, is in excellent agreement with subjective measurements.

13. The replica presents a possible means of studying the eye objectively.

14. The results of tipping of the cornea or the crystalline lens can be studied by their effect on the objective image of the replica.

15. Eventually, by means of the replica, it may be possible to separate the subjective from the objective factors of vision.

It is hoped that it will be possible to obtain fresh eyes, to examine them on a lens bench and to compare their optical images with those of the replica. This will be done photographically if possible.

The problems that remain to be solved are as follows:

To investigate the problem of the best optical image in an eye, and find out if it falls on the fovea or on some other spot, such as the end of the optic axis of Helmholtz.

To study the effect of the angle alpha on the formation of the optical image in the eye.

To determine whether or not the tipping of the crystalline lens has an appreciable effect on the image.

To replace the front surface of the replica by an aspheric surface representing more nearly the true shape of the front surface of the cornea, and to study the effect of this surface on the aberrations of the image formed by the spherical surfaces.

To replace the front surface of the replica by astigmatic, myopic and hyperopic surfaces to approximate more nearly the refraction of individual eyes, and to study the retinal images of these eyes.

To measure the angles of an eye by means of the ophthalmophakometer; then to set and adjust the replica until it gives the same images of Purkinje.

To examine the image formed in the previous case, and to compare its properties with the subjective readings for the individual eye.

To photograph the images of the replicas of three such individual eyes and to compare their objective impressions of the same thing.

To determine if the differences in their objective impressions are in agreement with the differences in their subjective impressions.

To determine whether the distortion of an eye is entirely geometric or partly psychologic.

To investigate thoroughly the coma of Tscherning's schematic eye by triangulation of rays which form the complete pencil of light entering the eye.

FORMULAS

Cardinal Points of a Lens System.—The English convention of signs is used in this part of the work, i. e., distances measured with incident light are minus, and against incident light are plus. These distances are measured from the pole of a surface and from the principal planes of a lens.

The cardinal points of a lens system are the two focal lengths, the two principal points, and the two nodal points.

The first focal point is the point at which an incident ray must cross the optic axis in order to emerge from the system in a direction parallel to the axis. The second focal point is the point at which an emergent ray, which was originally parallel to the axis, crosses the axis.

The second principal point is the image of the first; that is, the principal points are a pair of conjugate points on the optic axis, so that a ray which is aimed at the first principal point, before refraction, will issue from the system along a straight line which passes through the second principal point. The principal planes of the system are the pair of conjugate planes perpendicular to the axis at the principal points; and the characteristic property of these planes is that an object lying in the first principal plane is reproduced by an image in the second principal plane which has exactly the same dimensions as the object and is oriented in the same way. The principal points are the only pair of conjugate points of the system for which this condition is satisfied.

The two nodal points are likewise a pair of conjugate points on the axis, characterized in this case by the fact that an incident ray which is directed to the first nodal point will emerge from the system in the same direction along a line which passes through the second nodal point.

The distance of the first principal point from the first focal point is called the first focal length. The second focal length is defined as the distance of the second focal point from the second principal point.[54]

The following method was used for computing the cardinal points.[55]

(1) A single lens in homogeneous mediums:

$$f_1 = \frac{-R}{N-1} \qquad f_2 = \frac{RN}{N-1}$$

$$F_2 = P_2F_2 = \frac{-N\ f_1'\ f_2''}{N(f_2'' - f_1')\ t}$$

$$\text{alpha} = HP_1 = \frac{t\ f_1'}{\Delta_1}$$

$$\text{beta} = QP_1 = \frac{t\ f_2''}{\Delta_1}$$

(f_1 represents the anterior focal length of the first surface; f_2, the posterior focal length of the second surface; R, the radius of curvature; N, the relative index [N_2/N_1], and t, the thickness of the lens.)

(2) A thick lens in nonhomogeneous mediums:

$$F_1 = P_1F_1 = \frac{N_1N_2\ f_1'\ f_2''}{\Delta_2} \qquad F_2 = P_2F_2 = \frac{-N_2N_2\ f_1'\ f_2''}{\Delta_2}$$

$$\text{alpha} = AP_1 = \frac{N_1N_2\ f_1'\ t}{\Delta_2} \qquad \text{beta} = BP_2 = \frac{N_1N_2\ f_2''\ t}{\Delta_2}$$

$$\Delta_2 = N_2(N_1f_2'' - N_2f_1') + N_1N_2t$$

(3) Two thick lenses—general (equivalent lens):[56]

$$F_A = P_1F_1 = \frac{N_1f_1'f_1''}{N_2f_1' + N_1(f_1'' - d)} \qquad \text{alpha} = P_1'P_3 = \frac{-N_1f_1'd}{\Delta_3}$$

$$F_P = P_2F_2 = \frac{N_2f_2''f_2'}{N_2(f_2' + d) + N_2f_2''} \qquad \text{beta} = P_2''P_2 = \frac{N_2f_2''d}{\Delta_4}$$

(d equals the optical separation or the distance from P_1 of the second lens to P_2 of the first lens.)

$$F_1N_1 = P_2F_2$$
$$P_1F_1 = F_2N_2$$[57]

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54. Helmholtz (footnote 10, p. 61).

55. Wilder: Lens Formulas, thesis for master's degree, University of Rochester, 1922.

56. Edser: Light for Students, New York, The Macmillan Company, p. 74.

57. Helmholtz (footnote 10, p. 62).

Triangulation.—The German convention of signs is used in this part of the work.[58] Triangulation is the process of running an optical ray through a lens system by the solution of triangles formed by the incident ray, the refracted ray and the optic axis.

Light is aimed at a given point located by the object distance as measured from the first surface (u_1). Light is assumed to come from the left. Surfaces concave to incident light are negative, and surfaces convex to incident light are positive. Object distances to the left are positive. Image distances to the right are positive.

The method of procedure is as follows (u equalling the object distance; v, the image distance; i, the angle of incidence; r, the angle of refraction; m, N_2/N_1; U, the angle that the incident ray makes with the optic axis, and V, the angle that the refracted ray makes with the optic axis):

1. For oblique rays.

$$(1)\ \frac{\sin i}{R+u}=\frac{\sin U}{R} \qquad (2)\ \sin i=\frac{(R+u)\ \mathrm{Sin}\ U}{R}$$

$$(3)\ \sin i = m \sin r \qquad (4)\ \text{Deviation} = U-(i-r) = V$$

$$(5)\ \frac{v-R}{\sin r}=\frac{R}{\sin V} \qquad (6)\ v-R=\frac{R \sin r}{\sin V}=-N$$

$$(7)\ v = R-N \qquad (8)\ s-v=u$$

2. For parallel rays, the entrance value equals F/2S in which S represents the speed of the lens.

3. The oblique thicknesses and separations may be computed in the usual manner.[58]

4. Determination of the focal lines.[59]

(1) Primary:

$$\frac{m \cos^2 r}{v'}=\frac{m \cos r}{R}-\frac{\cos i}{R}-\frac{\cos^2 i}{u}$$

(2) Secondary:

$$\frac{m}{v''}=\frac{m \cos r}{R}-\frac{\cos i}{R}-\frac{1}{u}$$

Aberrations.—1. Spherical aberration: If the edge ray cuts the optic axis nearer the lens than the equivalent focal length, it is negative and designated by a minus sign.

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58. Frederick: A System of Triangulation for the Determination of the Aberrations of a Lens, unpublished formulas used at Hawk Eye Works, Eastman Kodak Company, Rochester, N. Y., 1908.

59. Nutting: Outlines of Applied Optics, Philadelphia, P. Blakiston's Son & Company, p. 28.

2. Sine condition: The sine condition test, since it is made for parallel incident light, changes the usual ratio of sin u/sin u′ to the expression h/sin u′, equalling a constant or f_c'.[60]

3. Coma: Coma is arbitrarily measured by the sine condition minus the spherical aberration. The real test is the relative shapes of the spherical aberration and sine condition curves.[6]

4. Distortion: The distortion is measured by the Le Grange-Helmholtz equation:

$$N_1B_1 \tan a_1 = N_2B_2 \tan a_2,^{61} \text{ or, better,}$$

$$\frac{N_1B_1 \tan a_1}{N_2B_2 \tan a_2} = k$$

If the distortion is negative it is designated as barrel, and if positive as pincushion.

5. Astigmatism: The astigmatism is measured by the difference in millimeters between the primary focal line and the secondary focal line.[62] The primary focal line is tangential, while the secondary is radial.[63] If the position of the focal line is between the focal plane and the lens it is designated as negative.

6. The curvature of the field should be such that the best image, or the circle of least confusion, falls on the focal plane at all possible angles.[64]

7. The chromatic aberration is obtained by computing the back focus for different wave lengths.[65]

8. Lateral chromatism is tested usually by the relative position of the principal points for the different wave lengths.[6]

9. The focal lengths, back focus and front focus, were checked by running an axial ray; for if one lets the angle of incidence equal zero, then i equals O, r equals O, and the formulas for the primary and secondary focal lines become

$$\frac{N_2}{v} = \frac{N_2 - N_1}{R} - \frac{N_1}{u}$$

and from this the back focus is obtained at once. If the direction of light is reversed the front focus is obtained. The equivalent focus is obtained by the following formula:

$$\frac{v_1 \times v_2 \times v_3 \ldots v_n}{u_2 \times u_3 \times u_4 \ldots u_n}^{66}$$

60. Lummer: Photographic Optics, New York, The Macmillan Company, p. 120.
61. Helmholtz (footnote 10, p. 74).
62. Jewell: Research Laboratory, Kodak Park, Eastman Kodak Company.
63. Lummer (footnote 60, p. 24).
64. Lummer (footnote 60, p. 25).
65. Nutting (footnote 59, p. 40).
66. Gleichen: Lehrbuch der geometrischen Optik, Leipzig, 1902. p. 90.

STENOSIS OF THE LACRIMAL DUCTS*

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Two years ago at a meeting of this society I outlined my technic in the treatment for dacrocystitis. While the number of patients treated is not large, the results have been so satisfactory that I wish to report not only the end-results but other points of interest.

Stenosis of the lacrimal ducts may be either congenital or acquired. These two forms are again divided into simple stenosis and stenosis with an infection of the lacrimal sac. The infections may be acute or chronic. There are also stenoses resulting from malignancy, traumatism and other conditions, but the latter types are not considered in this paper.

Congenital stenosis is accounted for by a failure of the central cells to degenerate and be removed from the cylinder in late prenatal life, and the child is born with an imperforate duct. The treatment for this condition consists of forcing an opening by passing a probe. Instead of passing the probe through a slit canaliculus, I make an opening directly into the sac just below the ligament of the orbicularis and pass the probe through this opening. This method has the advantage of leaving the canaliculus intact, of allowing the use of a larger probe and of lessening the danger of making a false passage. It also facilitates the proper cleansing of the sac and the application of any antiseptic desired to the mucous lining. The probing seldom needs to be repeated.

In a discussion of a paper read at the meeting of the American Academy of Ophthalmology and Oto-Laryngology at Detroit in 1927 Dr. Allen Greenwood said, "I want to make it emphatic, as I have every time I have spoken on this subject, that infants should not be probed." He gave as the reason for his position, that the operation requires an accurate knowledge of what one is going to do, and especially a knowledge of the direction of the lacrimal passages. It is true that the ducts of infants do not have the same direction as those of the adult, yet my observation is that the ducts do not vary in direction, one from another, more than do those of adults. If the operator has the sense of touch with a probe that he should have he will not cause trouble, and if the probing is done through an opening directly into the sac he should encounter no difficulties. I see no excuse for abandoning this treatment when it gives 100 per cent good results.

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* Submitted for publication, April 15, 1929.

* Read at the meeting of the Omaha-Council Bluffs Eye and Ear Society, March 20, 1929.

The treatment for acquired stenosis with or without a dacrocystitis by the Bowman method—that of passing small probes by way of the canaliculi—has been largely, and should be wholly, abandoned, as it yields only from 5 to 10 per cent cures. The treatment with the larger probes of Theobald and Ziegler gives better results, but not enough better to warrant a continuance, especially when a dacrocystitis is present.

Treatment by extirpation or obliteration does away with the infected sac and the danger to the eye from that infection, but the annoying epiphora remains to a greater or lesser degree.

In the last eight or ten years much work has been done, mostly by rhinologists, to perfect an operation the object of which is to provide drainage from the sac directly into the nose. This objective is attained by an external approach as by the Toti-Mosher operation, or an intranasal approach as by the Halle-West operation. The result desired by each of these methods is to secure a large opening from the sac directly into the nasal cavity at the anterior end of the middle turbinate body. Cures in from 60 to 90 per cent of the cases are reported by the operators using these methods.

In spite of the large percentage of cures reported, there are objections to these operations: 1. I cannot think it a safe procedure to conduct pus from the lacrimal sac directly into the nares in the region of the ethmoid cells, and especially if some of the anterior cells have been broken down as is usually done. There is also danger of infection of the maxillary antrum if pus is deposited in proximity to its ostium. 2. There is too much danger that infectious material might be forced from the nares into the eye, with so short and direct a connecting passage. 3. Another objection, which is not vital, is that of giving over a part of ophthalmic surgery to the rhinologist.

TECHNIC

I shall review the technic of my operation. If pressure on the sac forces its content through the canaliculi, one knows that they are patulous. If this cannot be done, a no. 4 Bowman probe is passed into the sac to make certain that the canaliculi are not closed. Next, after the tissues have been anesthetized, an incision is made into the sac. The cut extends from the ligamentum orbicularis down to the lower border of the lacrimal groove. The opening is about 7 mm. long. There is no necessity of cutting the ligament of the orbicularis, and it should not be done. Through the opening in the sac the content is washed out, and a 2 per cent solution of silver nitrate is used on a swab in order to wipe thoroughly the entire mucous lining. The sac is then packed with a wick dampened with a 10 per cent solution of cocaine with a drop of epinephrine added. In five or ten minutes the anesthesia is complete, and the probes are passed through the duct. I begin with a 2.5 mm. probe and follow by 3, 3.5 and 4 mm. probes. One cannot use all sizes in each case. A 4 mm. probe can be passed in some cases and a 3.5 mm. in most of them. I found one duct which would not admit a probe larger than

2.5 mm. After the duct has been dilated, a 2.5 mm. silver drainage tube is introduced and the dressing applied. At first I used a silver wire, but I find that a tube keeps the sac drained and is much more efficient. At intervals of two or three days, the sac is treated through the incision and the tube and dressing are replaced. When the sac is cured of infection and the duct is well dilated, the incision in the skin is allowed to close, which it does in two or three days without leaving a visible scar. From four to six visits are required, as a rule, to complete a cure.

Following this technic, I have treated nineteen patients. Of this number, Mrs. R., who had a mucocele, contracted smallpox before the

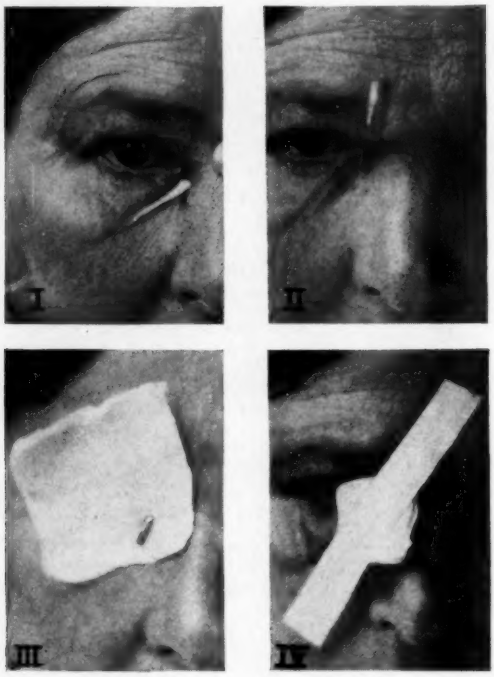

Fig. 1.—Showing implements used and technic of operation in stenosis of the lacrimal ducts: *I*, applicator for wiping out the sac; *II*, no. 16 (4 mm.) probe in the nasal duct; *III* and *IV*, method of dressing with 2.5 mm. silver drainage tube in the nasal duct.

treatment was completed, and I have not seen her since. Mrs. L., a woman of nervous temperament, after three treatments refused to continue them, and I removed the sac. Two patients, because of faulty judgment on my part, were discharged too soon. I reopened the sacs and renewed the treatment; in each case, cure resulted. Of the nineteen cases, cure resulted in seventeen, while treatment was not completed in two.

The percentage of cures in this small number of cases is as good as that from any other method of treatment. The infection of the sac was cured and the drainage reestablished through the normal passages, which is a distinct advantage over the Toti-Mosher or the Halle-West method. If the stenosis is caused by an infection from the nose, as a majority of physicians seem to believe, and at the time one relieves the stenosis the nose is in good condition, why should one expect a reinfection? If the stenosis is caused by an infection in the sac and that infection is cured and drainage is reestablished, there should be no return of the trouble. If there has been a return in any of my cases, I am not aware of it.

To effect a cure one must get rid of the infection in the sac. This can be done by the open method. To rid the sac of infection is tedious and next to impossible unless it is treated as an open cavity.

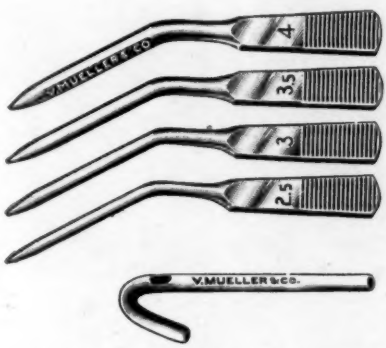

Fig. 2.—Ziegler probes cut in two and the 2.5 mm. silver drainage tube.

It is easier to cut directly into the sac than to slit the canaliculus properly. The large drainage tube can be used through the incision in the skin but not satisfactorily through the canaliculus. The canaliculi should not be slit except in those rare cases when it is necessary to perform curettage for tracoma, streptothricosis, etc., and then they should be allowed to close at once.

Free drainage is reestablished by the use of large probes followed by the insertion of the large silver tube which is left in the duct between dressings. Through a 7 mm. opening into the sac, one is able to study the lacrimal duct in a person as readily as in a cadaver. In all my cases except two the stenosis was at the upper end of the bony duct. In the two exceptions, the stenosis was about 1 cm. from the upper end.

The caliber of the ducts lined with mucous membrane is measured in millimeters by the size of the probe which can be introduced. When as large a probe as will enter the duct is passed, the handle can be moved forward and back and from side to side. The part of the probe which is

in contact with the upper end of the duct does not move, showing that the duct is of larger caliber at the lower end than at the outlet of the sac.

An ophthalmologist, with other accomplishments, should have a mechanical turn of mind. Such a one must see the advantage in this method. There is a closed cavity filled with pus, and the rational way to treat such a cavity is to convert it into an open cavity. Dilation and treatment of a lacrimal duct can surely be more easily and accurately accomplished when there is no tissue to go through before reaching the duct.

SYMPATHETIC OPHTHALMIA

REPORT OF A CASE OCCURRING FORTY-EIGHT YEARS AFTER INJURY *

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In a large majority of cases, sympathetic disease develops from two to eight weeks after a penetrating wound in the ciliary region, particularly when the iris, the ciliary body or the capsule of the lens has been lacerated. Reports have appeared of longer intervals before the disease manifested itself, especially when intra-ocular foreign bodies had been retained. When an injured eye has been free from inflammatory reaction for months or years, the subsequent appearance of inflammation in the second eye without a history of flare-ups or attacks of pain in the injured eye should be regarded as an independent condition, probably of constitutional origin and therefore not directly related to sympathetic ophthalmia. Schirmer,[1] Vignaux,[2] Sulzer,[3] Weeks [4] and Knapp [5] have reported cases as sympathetic ophthalmia in which the condition occurred fifteen, twenty-eight, thirty-seven, forty-two and forty-five years, respectively, after trauma.

The case reported here is presented because the diagnosis is substantiated by the patient's history, the clinical course and the microscopic sections of the enucleated eye and iris tissue excised from the sympathizing eye.

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* Submitted for publication, March 3, 1929.

* Read before the New York Academy of Medicine, Section of Ophthalmology, February, 1929.

1. Schirmer, Otto: Klinische und pathologische anatomische Untersuchungen zur Pathogenese der sympathischen Augenentzündung, Arch. f. Ophth. **38**:95, 1892.

2. Vignaux, J. C.: De l'ophthalmie sympathique et specialement de son traitement par l'enucleation, Librairie, J. B. Bailliere et fils, Paris, 1877.

3. Sulzer, D.: Ophthalmie sympathique 37 ans apres le traumatisme, Ann. d'ocul. **137**:141, 1907.

4. Weeks, J. E.: A Case of Sympathetic Ophthalmia Occurring Forty-Two Years After Loss of Exciting Eye: Recovery, New York Eye & Ear Infirm. Rep. **2**:30, 1894.

5. Knapp, Herman: Ueber Knochenbildung im Auge, Arch. f. Augenh. **2**:133, 1871.

Whitening of the eyelashes (poliosis), seen in rare instances of sympathetic ophthalmia, was also observed in this case. Its occurrence has been noted by Jacobi,[6] Nettleship,[7] Tay [8] and Cramer.[9]

REPORT OF A CASE

History.—H. K., aged 60, was first examined on Nov. 3, 1926, when he complained of severe pain in the left eye which radiated to the frontal region. The left eye had been injected and slightly painful during the past few months. Forty-eight years previously, at the age of 12, he sustained a penetrating wound when a fellow student thrust a penpoint into the left globe. He was treated in a hospital in Odessa, Russia, but there was no restoration of vision, and at the age of 21 he was rejected for army service because of blindness in the left eye. The eye did not cause any trouble until thirty years before this examination, when he suffered an attack of pain and redness, and an enucleation was advised by Dr. Paine of New York. He has suffered from periodic attacks of pain of moderate severity during the past thirty years.

Diagnosis.—The left eye showed iridocyclitis following an old perforating wound, which was complicated by cataract, old exudate in the vitreous, secondary glaucoma and beginning ciliary staphyloma superiorly. There was no perception of light. Vision was 15/20 in the right eye, with combined hyperopic astigmatic correction. The pupillary reaction was sluggish and incomplete, and slight ciliary injection was present. Dilation revealed five posterior synechiae inferiorly. The upper half of the pupil was dilated fully and equally. Examination with the slit-lamp revealed old deposits of pigment on the anterior capsule in the lower half. There was ophthalmoscopic evidence of retinal arteriosclerosis. Chronic, inactive iritis was diagnosed.

Course.—The patient was admitted to the Post-Graduate Hospital, where the results of the physical examination were negative except for a suggestion of a healed lesion at the apex of the left lung. On Nov. 4, 1926, enucleation of the left eye was performed, with gold ball implantation. Following an uneventful recovery, the patient was discharged one week after operation.

Nine days after enucleation, the patient complained of transient cloudiness in the right eye, and atropine was prescribed, which fully dilated the pupil in the temporal half. Treatment for plastic iritis was successful, atropine, dry heat, potassium iodide and acetylsalicylic acid being used. On December 14, a second attack of iritis developed; the condition responded to treatment in two weeks. There was no increase in the intra-ocular tension. There was diminution of vision from 15/20 to 15/30. On Jan. 2, 1927 (eight and one-half weeks after enucleation of the left eye), there was a recurrence of right-sided pain with a fresh increase in the amount of plastic exudate on the capsule of the lens. On January 24, the

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6. Jacobi, Joseph: Vorzeitige und acute Entfarbung der Wimpern beschrankt auf die Lider eines sympathisch erkrankten Auges, Klin. Monatsbl. f. Augenh. **12**:153, 1874,

7. Nettleship, Edward: A Case of Sympathetic Ophthalmia with Whitening of the Eyelashes, Tr. Ophth. Soc. U. Kingdom **4**:38, 1884.

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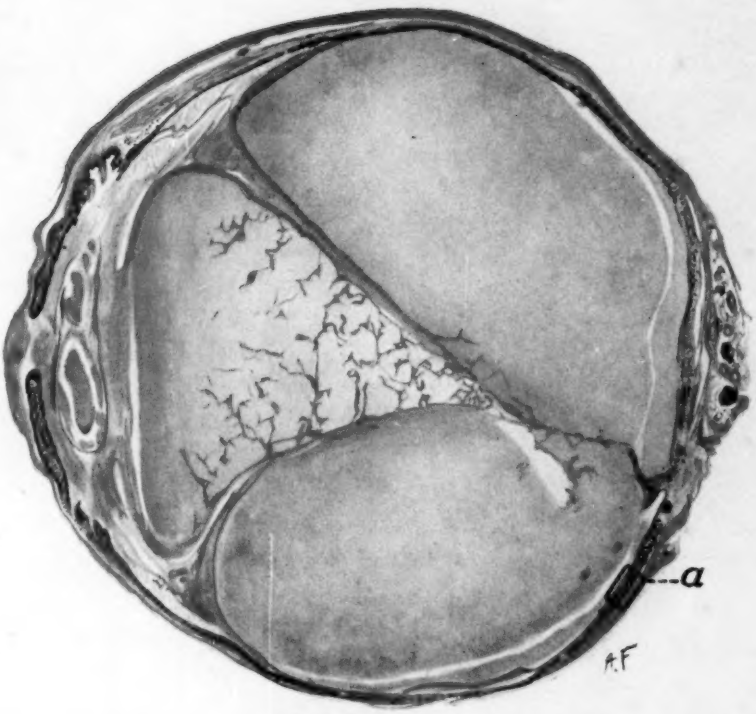

Fig. 1.—Sympathetic ophthalmia after a wound of the cornea. The iris, ciliary body and chorioid show marked lymphocytic infiltration. The anterior and posterior chambers are filled with exudate. The lens is wounded and there is a cyclitic membrane. The retina is detached with subretinal exudation. Foci of lymphocytes extend through the sclera about the posterior ciliary vessels.

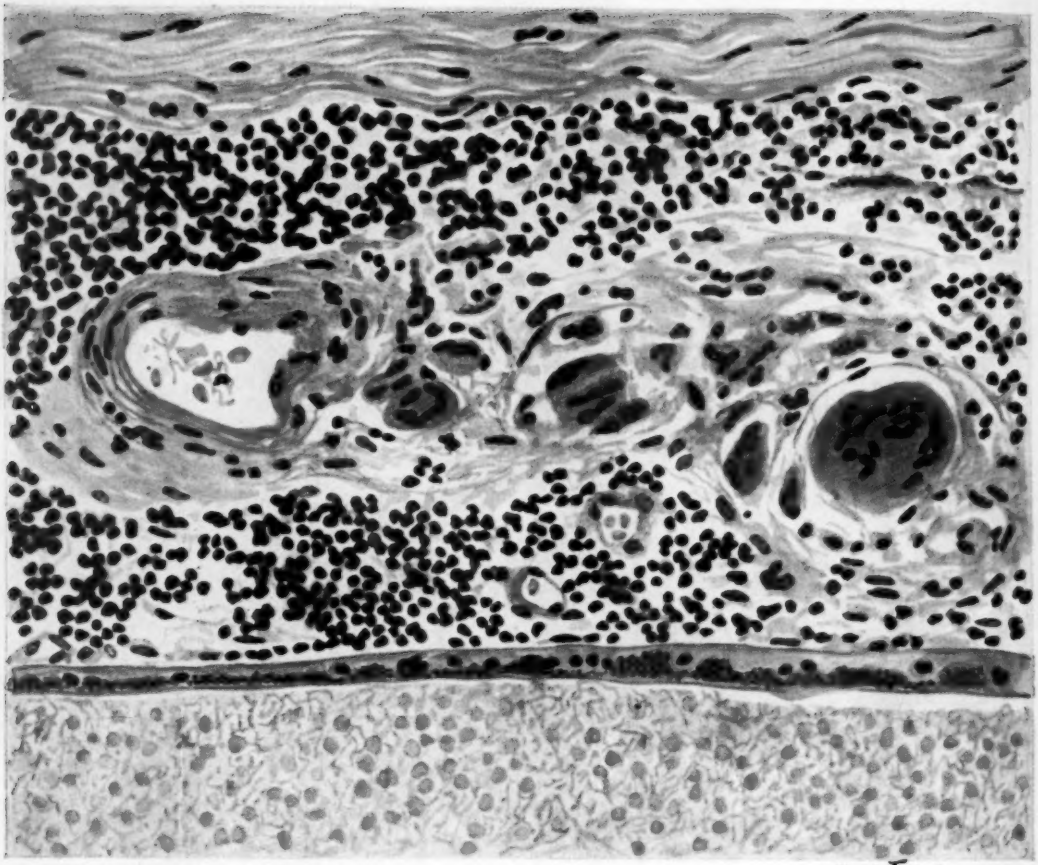

Fig. 2.—High magnification of *a* in figure 1. The much thickened chorioid is diffusely infiltrated by lymphocytes and fewer plasma cells. The giant cells are surrounded by epithelioid cells.

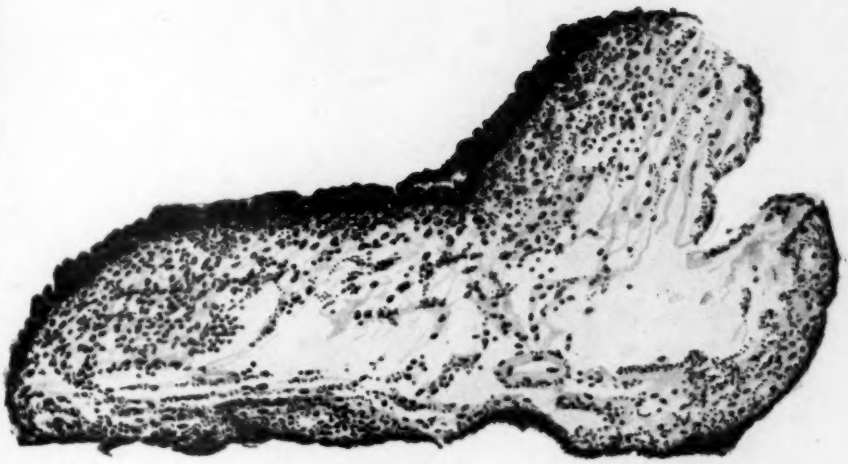

Fig. 3.—Sympathetic iritis in the sympathizing eye. The excised portion of iris shows marked infiltration by lymphocytes, very typical of the early stage in the sympathizing eye. The spaces represent iris crypts.

presence of fresh fatty deposits on Descemet's membrane was noted, and one week later there was an attack of acute pain. On examination, the cornea appeared cloudy; the bulbar conjunctiva was markedly injected, and there was ciliary tenderness, marked superiorly. Tension with Schiotz' tonometer was 70; after massage, 60. Vision before tonometric examination was 1/200; after examination, application of myotics and massage, 15/70. The patient was readmitted at once (January 31) to the Post-Graduate Hospital and responded to medical treatment for glaucoma. About this time the pathologic report on the enucleated eye was submitted by Dr. Louise H. Meeker, with a diagnosis of sympathetic ophthalmia. On receipt of the diagnosis, systemic treatment was introduced to counteract the sympathetic iridocyclitis in the right eye with 130 grains of acetylsalicylic acid during the day (10 grains hourly) to the toxic point. On February 8, the patient was discharged from the hospital. Vision was 15/50 and the tension 30. The cornea was clear and of normal luster, and the fundus was seen, although it was blurred by exudation on the anterior capsule of the lens. Treatment given at the office was followed by improvement of vision to 15/40 plus.

On February 24, there was a recurrence of pain and slight cloudiness of the cornea; the tension was 42.5, and was reduced to 30 by the administration of physostigmine (eserine). Vision was 15/100. Examination with the slit-lamp showed the presence of numerous deposits on the cornea, which was uniformly cloudy. The pupillary space nasally appeared to be glued off by débris of degenerated pigment and plastic exudation. Temporally, the pupillary space showed pigment in exudate less dense than that seen nasally. On February 26, the patient was referred to another ophthalmologist for consultation with the diagnosis of sympathetic ophthalmia. In his opinion, however, the iris did not show the flat adhesions usually seen in sympathetic ophthalmia, and it was his suggestion that a peripheric iridectomy be performed and that injections of diphtheria antitoxin be given postoperatively. His diagnosis was plastic iritis and not sympathetic ophthalmia. The patient was also examined by a second consultant who offered the additional suggestion that foreign protein, preferably typhoid, be injected before operation. At a conference of the ophthalmologic staff of the Post-Graduate Hospital, the general opinion was that medical treatment, rather than surgical intervention, be continued. On March 1, iridectomy was performed, including the iris root, and the specimen was sent to the laboratory. Three days after the operation, Verhoeff's treatment for sympathetic uveitis with diphtheria antitoxin was given. The intradermal skin test for diphtheria antitoxin was strongly positive. Treatment was begun with graduated desensitizing doses hourly of 0.1, 0.2, 0.5, 1, 1.5 and 2 cc., after which the balance of 12,200 units was injected. On March 6, 7, 8 and 9, 15,000 units was injected, and on March 11, 13 and 15, 18,000 units.

Postoperative Course.—On March 3, the fingers could be perceived at a distance of 2 feet. Plastic exudate filled the lower half of the coloboma of the iris. On March 6, vision was 4/70; the pupil was dilated more by 5 per cent atropine; the plastic exudate was thinner in the area of the coloboma, and the cornea and the iris appeared clearer. Pathologic examination of the excised iris showed changes of sympathetic ophthalmia, less marked than in the enucleated eye. The patient was discharged from the hospital on March 16, with a vision of 10/200. On March 22, he was examined by Dr. Arnold Knapp and Dr. F. H. Verhoeff, who also examined the slides of sections of the injured eye and verified the pathologic report. General treatment for sympathetic ophthalmia was continued at the patient's home, without causing further improvement in vision. On May 10, two months after iridectomy, more synechiae developed in the pupillary space, the coloboma was

covered by exudation and iris bombé was noted. Vision was 6/200 and tension 22.5. There was beginning poliosis or blanching of the lashes of the right upper lid. On Jan. 11, 1928, there was further visual reduction to perception of light in the temporal field only; it was faulty in the nasal field. Occlusio pupillae, seclusio pupillae and iris bombé were noted but the tension was not increased. The cilia of the upper lid, except a few in the middle third, were now pure white.

The patient was last examined on Feb. 4, 1929. There was extreme blanching of the skin of the right eyelids superiorly to the eyebrow, below to the orbital ridge, nasally to the canthus and temporally beyond the orbital margin. All cilia of both lids were colorless, except a small tuft of sixteen eyelashes in the middle third of the upper lid. The hairs of the eyebrow were uniformly black. The cilia of the left eyelids were of normal blackness throughout. The hairs of the left eyebrow were about one-fourth gray.

Summary.—The patient's left eye, blinded in youth after a penetrating wound, suffered recurrent attacks of pain and became stony hard; enucleation was performed forty-eight years afterward.

Nine days following enucleation of the left eye, which showed old posterior synechiae, there developed plastic iritis, which responded to treatment. One month after this onset a second attack followed, and nineteen days thereafter a third attack. Eleven weeks after enucleation, a fourth outbreak of plastic iritis was complicated by increased intra-ocular pressure (70 mm. of mercury Schiotz) necessitating hospitalization. Pathologic sections of the enucleated eye revealed changes typical of sympathetic ophthalmia. The glaucomatous pressure in the right eye was reduced by miotics and acetylsalicylic acid in massive doses to the point of toxicity. Despite continuous observation and treatment for six weeks, there was a return of tension (42.5 mm. of mercury) and further visual loss. Peripheric iridectomy followed by injections of diphtheria antitoxin and salicylates failed to arrest the rapid reduction in vision from 15/40 to 10/200; finally amaurosis occurred ten months after operation. Microscopic examination of the excised iris showed a pathologic process similar to that found in the exciting eye, but to a lesser degree.

Two months after iridectomy on the sympathizing eye, depigmentation of normally black cilia and the skin of the right upper lid resulted, with eventual total blanching of the cilia and pallor of the entire skin covering of both right eyelids. The cilia and skin of the left lids remained unchanged.

Report of Pathologic Examination of the Left Eye, Post-Graduate Hospital (by Dr. L. H. Meeker).—The specimen was taken on Nov. 4, 1926, and the report was made on March 1, 1927.

Gross Examination: The eye measured 27 by 24 by 23 mm. The cornea was opaque. On section, the eye was filled everywhere with a blood-tinged albuminous exudate.

Microscopic Examination: The epithelium of the cornea was irregular in thickness, and between it and Bowman's membrane there was a layer of scar tissue and blood vessels, that is, pannus degenerativus. In the deeper stroma, a few blood vessels extended inward from the limbus for several millimeters. Aggregations of plasma cells and lymphocytes surrounded the anterior ciliary vessels. Schlemm's canal and the meshwork of Fontana were obliterated by compression and infiltration.

The anterior and posterior chambers were filled with a connective tissue membrane. This membrane extended into the vitreous, incorporating the iris, ciliary body, remnants of the lens and the hardly recognizable retina. The cyclitic portion of the membrane had pulled the retina forward and obliterated all landmarks. In

the irregular loops and spaces thus formed there was a fairly recent serosanguineous exudate, and beneath the retinal shreds a similar exudate filled the spaces between the choroid and retina. Smaller spaces everywhere contained fresh blood. The retinal elements were disintegrated beyond recognition and bands of scar tissue seemed to connect shadowy folds suggesting a former detached retina. Some of the bands of scar tissue extended from the retina to the choroid.

The whole uveal tract presented a severe and everywhere similar inflammatory reaction. It was thickened by a diffuse infiltration of lymphocytes and plasma cells, and scattered throughout this exudate were many large and small foci of epithelioid cells with occasional cells suggesting Langhans' giant cells. This inflammatory exudate surrounded the ciliary vessels and vortex veins and passed through the emissaria to the episclera with dense foci about the circle of Zinn vessels. The reaction throughout the uveal tract was typical of sympathetic ophthalmia.

The lens remnants were enclosed in the folded capsule and many spindle cells spread beneath the capsule and among the lens fibers. A few giant cells and many endothelial cells also clung to the broken lens fibers. Blood vessels beneath and on the capsule contained abundant polymorphonuclear leukocytes, and extravasations of blood surrounded them.

Diagnosis: Sympathetic ophthalmia, recent multiple hemorrhages, iridocyclitis plastica, detached and degenerated retina, secondary glaucoma, complicated cataract, reaction to lens protein (?) and pannus degenerativus were diagnosed.

Pathologic Report of Examination of the Iris Tissue of the Right Eye (Dr. Meeker).—The specimen was taken on March 1, 1927, and the report was made on March 5, 1927.

Microscopic Examination: The minute fragment of the iris showed diffuse infiltration of the stroma by small lymphocytes and occasional plasma cells. Certain mononuclear cells in the iris stroma suggested epithelioid cells. Pigment granules were scattered throughout and the pigment epithelium showed proliferation.

Diagnosis: Chronic iritis, resembling the reaction in the enucleated eye, but less severe, was diagnosed. Sympathetic ophthalmia was suggested.

MANAGEMENT OF SIMPLE GLAUCOMA

A CLINICAL REPORT *

WALTER R. PARKER, M.D., Sc.D.
DETROIT

The subject of simple glaucoma is one about which so much has been written that it is with some temerity I even venture to make a suggestion concerning its management. I shall, however, begin by presenting the results obtained from the use of epinephrine and a proprietary epinephrine substitute (Link's Glaucosan) in a limited number of cases, and give the comparative results obtained following iridectomy and the trephine operation.

CLASSIFICATION AFTER TREATMENT

If ophthalmologists were to make a post-treatment classification of patients assumed to have simple glaucoma, the following grouping might be possible:

1. The type in whom spontaneous recovery occurs.
2. The type in whom the condition will be arrested by general treatment and the use of miotics.
3. The type in whom it will be arrested by proper surgical intervention.
4. The type in whom the condition will progress in spite of all efforts.

Type 1.—This type includes patients with swollen lenses in the initial formation of cataract and with cases of low-grade uveitis. The condition should probably be classed as secondary glaucoma, although when the patient was first examined this may not have been possible. The condition of the lenses should play a part in the early diagnosis of glaucoma.

Type 2.—The second group includes patients influenced by general treatment and the local use of miotics and epinephrine and an epinephrine substitute (Glaucosan). All these patients were given general and local treatment without regard to the stage of the disease when they came under observation. Those treated by physostigmine (eserine) and pilocarpin will not be considered in this report.

Twelve patients with simple glaucoma were treated by subconjunctival injections of epinephrine. While the tension was reduced temporarily in the majority of the patients, the treatment proved beneficial in only one.

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* Submitted for publication, April 6, 1929.

* Read at the New York Academy of Medicine, Section of Ophthalmology, March 18, 1929.

An epinephrine substitute (Link's Glaucosan) was administered, after the method described by Hamburger, to nine patients with simple glaucoma, and in none was the tension satisfactorily reduced. In one the tension increased. It was also employed without success on two patients with secondary glaucoma with old posterior adhesions.

I shall mention the results obtained from the use of the same epinephrine substitute in patients with iritis without glaucoma. Last fall Dr. Ellett read a paper in Chicago on the use of this drug. After mentioning the unfavorable results experienced in simple glaucoma, he reported eleven cases of iritis with posterior adhesions in which the patients had been benefited by the use of the drug. This paper prompted me to try the preparation, with the following results: In twelve patients with iritis of long standing, there was no improvement; in one patient with sympathetic ophthalmia, there was no improvement; in six patients with acute iritis with posterior adhesions, all the adhesions were torn loose after the use of atropine had failed. One patient, with a condition of two weeks' duration, did not receive treatment. A single instillation dilated the pupil in fifteen minutes. Doses of epinephrine substitute (Glaucosan) were not repeated, but were followed by the use of atropine. Doubtless, some of the patients would have responded to the continued use of atropine.

I have had no experience with a second proprietary epinephrine substitute, Amin-Glaucosan.

Type 3.—This group includes those patients in whom the tension was arrested by proper surgical intervention.

A few clinical facts stand out in the experience of all who have dealt surgically with glaucoma. One significant fact is that there is no direct relation between the depth of the anterior chamber and the tension of the globe. Also, it may be stated that an iridectomy arrests the tension in some patients, has no effect on others and makes a few worse. The probabilities are that there is no operative procedure that is best suited to all patients with simple glaucoma.

The final test of any procedure for the relief of glaucoma must include the remote effect that it may have on the tension and integrity of the globe, and on its physiologic function. The final result in the individual patient depends on the type and stage of the disease at the time of treatment. As the majority of the patients referred to here were subjected to a trephine operation after vision had become seriously impaired, comparison can be made only as to the effect of the operation on the tension and integrity of the globe.

The results were classified as good when the tension was reduced to between 5 and 25 mm. according to the Schiötz tonometer, and no demonstrable complications followed the operation that would have

resulted in the diminution of visual acuity. Only by this surgical classification could all the patients operated on be included in the report.

The Lagrane operation and the iridotasis operation were resorted to in a limited number of patients, and from the results obtained no serious argument could be raised against either procedure. The patients were too few, however, to justify a comparative study.

A comparative study is made of the results obtained by iridectomy and the sclerocorneal trephine operation. The selection of patients for iridectomy was based on the following conditions: (1) a moderately deep anterior chamber, (2) fields not markedly contracted, (3) iris not atrophic, and (4) cases showing variable tension after the use of miotics (suggestion of Dr. Knapp).

The rest of the patients, data concerning whom are included in this report, were subjected to the sclerocorneal trephine operation.

Comparative Results Obtained from Iridectomy and Trephine Operations in Simple Glaucoma

	Number	Good Results
Iridectomy:		
Previously reported	19	13 = 72%
Additional	29	17 = 58%
Total	48	30 = 62%
Trephine:		
Previously reported	41	31 = 75%
Additional	73	56 = 76%
Total	114	87 = 76%

In the cases previously reported, a "follow-up" revealed the fact that about 17 per cent of the cases of simple glaucoma first classified under "good results" had to be transferred to a list of "poor results" or "failures." The number of additional cases here reported includes results obtained during a period of time varying from two weeks to seven years.

In two cases in which iridectomy was performed, classified as "poor" and not included in this report, the tension was 27 and 29, respectively. Also, two in which the tension was not increased on examination with the fingers, and in which the tension was not taken with the tonometer, are not included. One case in which trephining was done, not included, showed a tension of 27, and three in which the tension was not increased on examination with the fingers, but in which the tension was not taken with the tonometer, were also omitted.

At the same operative period eight patients had a trephine operation performed on one eye and an iridectomy on the other. In five, the results were good in both eyes. In two patients on whom iridectomy failed, a trephine operation was performed later with satisfactory results in one and failure in the other.

In all there were nine patients on whom iridectomy failed to reduce the tension and on whom the trephine operation was performed, with four failures and five good results. In those previously reported, there were six cases with five good results. This makes a total of fifteen patients with ten good results, or 66.6 per cent.

By reference to the table it will be noted that in sixty-three of the patients on whom iridectomy was performed the tension was reduced to between 5 and 25 mm. according to the Schiötz tonometer; that 76 per cent of the patients on whom a trephine operation was performed gave equally good results, and, finally, that on fifteen a trephine operation was performed after an iridectomy had failed, with good results in 66 per cent.

I cannot help feeling that if a proper selection of patients having simple glaucoma could be made for certain operative procedures, the average results would be better than when all are subjected to any one procedure. I am not sure that the selection here offered is the best that can be worked out, but it afforded a more definite method of approach, and the average results in my hands were better than when no attempt at a selection was made.

Type 4.—In this group are included those patients in whom the disease progresses in spite of all efforts. They may be classified in one of two groups: In the first group, the tension is reduced, but the atrophic process in the nerve continues. Fortunately, this group is small. The results are comparable to those of a perfect operation for cataract on a patient with optic atrophy; the surgical classification might show a perfect result, while the visual classification showed failure. In the second group, the tension remains high in spite of all operative procedures.

I shall speak about one patient illustrating this second group. In a girl, aged 18, with congenital syphilis, objects could be seen with the right eye and the vision in the left eye was 1/60; the tension in both eyes was from 40 to 60 mm. according to the Schiötz tonometer. Concentrically the field was contracted about 20 degrees, with a target of 10 mm. No central scotoma was demonstrable. In the course of two years an iridectomy, three trephine operations and two cyclodialyses were performed on the right eye, and an iridectomy, two trephine operations and one cyclodialysis on the left. The tension was reduced for a time after each operation, but it did not remain long below 30. At present, with the use of miotics, the tension remains between 30 and 40 mm. The remarkable fact is that the eyes are in nearly the same condition that they were before the surgical intervention. The only unusual feature was the fact that the subconjunctival tissue was very thick, more like that seen in the rabbit than that in the human being.

CONCLUSIONS

The average results from surgical treatment for simple glaucoma, if all stages of the disease are included, might be stated as follows: Sixty per cent of the patients operated on will have the tension satisfactorily reduced and the the integrity of the globe maintained; in 30 per cent, the condition will be uninfluenced by the operation, and 10 per cent may be made worse by the operative procedure.

The cases reported are from the ophthalmic clinic of the University of Michigan.

DISTURBANCES OF VISUAL FUNCTIONS IN CONCOMITANT CONVERGENT SQUINT

WITH SPECIAL REFERENCE TO RETINAL INCONGRUITY *

WILLIAM F. MONCREIFF, M.D.
CHICAGO

The most obvious feature of concomitant convergent squint is the deviation itself, but there are certain disturbances of the visual functions which are frequently present and which are of basic importance either in themselves or in their relation to the deviation. These disturbances comprise deficiency of the fusion faculty, amblyopia ex anopsia and retinal incongruity or abnormal correspondence.

FUSION DEFICIENCY

That at least the milder grades of fusion deficiency are functional is shown by the success of Worth [1] and others in developing normal binocular vision in these cases. While the anatomic existence and location of a fusion center are not yet established, Ohm [2] expressed the belief that a defect of fusion may be caused by a lack of connection between the vestibular centers and the descending central visual radiation fibers. In support of this hypothesis, he cited the observation of abnormalities of the induced vestibular nystagmus in cases of squint,† and also the high incidence of squint (42 per cent) which he found in cases of spontaneous ocular nystagmus. Such defects as these would seem to be organic and not subject to cure by functional training methods. Although a complete understanding of the nature of these defects of fusion must await the results of further investigation, it has been amply demonstrated from the clinical side by Worth, and subsequently confirmed by the experience of many others, that these defects are the primary or basic cause of squint, and that all other factors, such as the convergence excess of hyperopia, vertical imbalance, high-grade anisometropia, acute illness and convulsions, are secondary. These views of the etiology of squint are generally accepted; yet even recently other theories of the causation of squint have been offered, as for example by Wiedersheim,[4] and also by Doesschate,[5] who advocated Snellen's contention that abducens paresis, often bilateral, is always the cause of true convergent squint. In support of this idea, the latter cited

* Submitted for publication, April 20, 1929.
* Read at a meeting of the Chicago Ophthalmological Society, March 19, 1929.

† For the sake of brevity, the term "squint" is used throughout as synonymous with the expression "concomitant convergent squint."

cases in which there was inconstancy of the squint angle in different directions of fixation, difference between the primary and secondary deviation, subnormal abversion, past pointing and diplopia. This cause is undoubtedly responsible for a small proportion of the cases of squint, but surely not, as these authors contended, for all.

The group of cases in which the squinting eye has an organic lesion of the media or fundus is eliminated from consideration in this paper.

AMBLYOPIA EX ANOPSIA

Amblyopia ex anopsia, which may also be designated as acquired amblyopia, is the second important disturbance that has to be considered.

I shall analyze the situation which exists in the early period of squint concerning the relation between the images in the squinting and the fixing eyes. There is, of course, a shifting of all images toward the nasal side in the squinting eye, in accordance with the squint angle, but one needs to consider essentially only two areas in each eye, namely, the macular area and the eccentric area to the nasal side of the macula which receives the same image as the macula of the other eye. The phenomena concerning the images on these two areas in the squinting eye are of special interest and importance. The image on the macula of the squinting eye is not the same as that on the macula of the fixing eye, and this might give rise to some conflict between the two maculae, producing confusion, were it not that voluntary suppression of the image on the macula of the squinting eye quickly occurs. The image on the macula of the fixing eye is the same as that on the eccentric area of the squinting eye, and this combination produces diplopia. This diplopia may be abolished by exclusion or suppression of the eccentric image in the squinting eye, which was experimentally demonstrated by Sachs [6] in 1897. When this suppression mechanism occurs, it is at first voluntary, later subconscious, and, when prolonged continuously, leads to acquired amblyopia. A second mechanism by which these difficulties may be relieved is by the development of a new (abnormal) correspondence between the retinas, so that the eccentric retinal area in the squinting eye which receives the image of the object fixed by the other eye becomes the new center of orientation for the squinting eye. These changes are of course functional, and their proper location is undoubtedly the occipital cortex, but for the sake of simplicity they may be discussed as if they concerned only the retina.

A priori, one would expect the acquired amblyopia of squint to be characterized at least by a central scotoma for white and colors, and an eccentric scotoma of the same kind corresponding to the eccentric retinal area of the squinting eye previously referred to, these being the two areas in which exclusion or suppression chiefly occurs. Another view is that in acquired amblyopia there is impaired function of the entire

binocular part of the field of the squinting eye. The observations of Worth [1] and others concerning the nature and frequency of acquired and congenital amblyopia are of interest. Worth [1] said that in amblyopia ex anopsia the peripheral limits of the field are normal, and there is an absolute central scotoma which in extreme cases extends 15 or 20 degrees from the fixation point, with vision as low as bare light perception, and vision of counting fingers at 1 or 2 feet in the periphery. Peter [6a] said that the peripheral field limits are contracted, and that the central scotoma is relative, not absolute, in acquired amblyopia. Worth [1] described congenital amblyopia as occurring nearly always in eyes with a high compound hyperopic astigmatism, but rarely apart from squint. The central acuity is never less than 6/60, the peripheral form vision is normal up to within 20 degrees of the fixation point, the peripheral limits of the fields for form and colors are normal, the central color perception is normal and there is no scotoma. Of 193 patients with squint seen early in childhood and within a short time after the onset of the deviation, Worth [1] found congenital amblyopia in about 15 per cent. Among many thousands of patients with faulty refraction without squint, he found only twenty-three cases of amblyopia which he could regard as congenital.

Lloyd [7] believed that the amblyopia of squint is due to a pathologic cause in practically all cases, and he emphasized especially congenital syphilis and birth hemorrhages as causes of the visual defect. He expressed the belief that amblyopia ex anopsia is either rare or nonexistent, and showed that in cases which he designated so-called amblyopia ex anopsia the field defects found were of organic and not of functional origin. In these cases he found marked contraction of the peripheral limits of the fields for white and red, enlargement of the blind spot and small (from ½ to 1 degree) relative or absolute central defects for colors or even white, but never large central defects, even in eyes with the most marked amblyopia. He emphasized the finding of similar field defects of slight grade also in the fixing eye as further evidence of a pathologic basis for the changes. As the number of patients examined was not very large, and as none were examined prior to the age of 10 years, there is no direct proof that Lloyd had any real cases of amblyopia ex anopsia, so that the conflict of his observations and views with those of Worth may be more apparent than real.

Evans [8] has just published a study of the scotoma associated with strabismus, based on 100 cases, of which he selected sixteen as meeting certain criteria which he established. These patients all had an absolute central scotoma with a diameter of 2 degrees or a radius of 1 degree, on the average, in the amblyopic squinting eye, which had an average acuity of 6/22. The ages ranged from 9 to 50 years, the average being about 25, and all were cases of early onset (prior to the age of 4 years), and

neglected early care, none of the patients having had glasses prior to the age of 9 years. All were nonalternating and had cosmetic parallelism, attained practically spontaneously, except in one case in which tenotomy had been done. The author referred to the idea that the macula is not fully "educated" until the seventh year, and speculated that on this basis the eccentric zone 1 degree from the macula would require twenty years, on the average, to learn fixation, and he seemed to consider this the essential mechanism by which the squint angle diminished in these cases to a deviation of only from 1 to 3 degrees. He showed the normal acuity of the zone 1 degree from the macula to be 6/12, which is considerably higher than the average in these amblyopic eyes, but he had some explanations to account for the discrepancy. He offered the hypothesis that this central scotoma may be caused by arrested development of the macula, even in the stages continuing after birth, as an interruption of the "uncovering" process, but considered that this explanation on an organic basis need not exclude the possibility of removal of the defect by educational means. This line of reasoning, however, could scarcely apply to the development of acquired amblyopia in an eye which had previously acquired normal acuity.

The theory of amblyopia ex anopsia was universally accepted until 1871, when Schweigger [9] denied that the amblyopia of squint is ever acquired and asserted that it is always congenital. Among numerous authors who believe in amblyopia ex anopsia in squint some of the foremost are Donders,[10] Javal,[11] Albrecht von Graefe,[12] Leber,[13] Fuchs,[14] Schnabel,[15] Bielschowsky,[16] Worth [1] and Priestly Smith.[17] Among those who oppose the idea of amblyopia ex anopsia are some who, like Schweigger, deny its existence, while others, like Uhthoff,[18] began with the same point of view, but later conceded the existence of amblyopia ex anopsia in a small number of cases, none of which they had personally seen, but continued to insist that the amblyopia of the squinting eye is congenital in the vast majority of cases.

Other outstanding opponents of the theory of amblyopia ex anopsia include Snellen,[19] Silex,[20] Alfred Graefe,[21] Landolt,[22] Wadsworth,[23] Roosa,[24] de Schweinitz [25] and a few others. Silex [20] and other opponents have asserted that there are no definitely known examples of lowered visual acuity of the squinting eye which developed after the onset of the deviation, or, as Schweigger stated it, no one has ever proved that an eye which once saw well has become amblyopic after squinting. It is also stated by some of these authors that they have never seen satisfactory evidence of improvement of vision of a squinting eye after operation, and they attempt to discredit such reports by saying that the apparent improvement is due to mistakes in examination. Uhthoff [18] stated that he was never able to secure improvement of the acuity of the amblyopic eye even in children under 6 years of age. In 43 per cent of the

amblyopic eyes of his patients with squint, he found other anomalies that he regarded as congenital, among which he included, however, as the chief proportion, high ametropia. As further arguments against acquired and in favor of congenital amblyopia, he referred to certain families in which some of the members have amblyopia combined with squint, and others have amblyopia without squint; he found amblyopia without squint in 10 per cent of his cases. He also stated that amblyopia of the squinting eye is slight in some cases of early onset, while the amblyopia is marked in some cases with a late onset of the squint; further, that there are patients with nonalternating squint with equal and normal visual acuity in both eyes.

Wadsworth [23] quoted Theobald [26] as advancing the view that the progress of amblyopia ex anopsia stops when it has reached such a degree and kind as shall do away with the discomforts of diplopia. The truth of this statement may well be doubted, but Wadsworth used it as the basis for an argument against acquired amblyopia, saying that on this account the degree of visual loss should be approximately the same in all cases, or that it should have some relation to the duration of the squint.

The nonoccurrence of amblyopia in senile cataract has also been adduced by some authors as evidence against acquired amblyopia. The answer to this contention is obvious.

As I present the evidence in favor of amblyopia ex anopsia, the refutation of the arguments stated against its existence will become evident. To begin with, it cannot be emphasized too strongly that the development of this amblyopia, as well as its cure, is confined essentially to the early period of childhood, prior to the age of 6 or 7, and the younger the child, the more striking is this relationship. The time required for the development of acquired amblyopia to the point of loss of central fixation, in relation to the age of onset of the squint, is rather definitely stated by Worth [1] as follows: In constant unilateral squint beginning at the age of 6 or 8 months, the power of central fixation of the squinting eye is often lost in eight or ten weeks; with the onset of squint at the age of about 18 months, central fixation is usually not lost for five or six months; with the onset of squint at the age of 3 years, central fixation is not lost for a year, and when the onset of squint is as late as 6 years or older, he has never observed loss of central fixation.

The direct evidence for the existence of acquired amblyopia is of two main varieties: (1) that concerning the decline of vision of the squinting eye, and (2) that concerning the improvement of vision of the amblyopic (squinting) eye. In addition, there is some indirect or collateral evidence, which, though of secondary importance, is of considerable interest, and consists in the occurrence of amblyopia ex anopsia in conditions other than squint, as has been pointed out by several authors referred to by Luedde,[27] including Dufour,[28] von Hippel,[29] Leber [13] and

Martin.[30] These observations concern the well known development of this amblyopia in some patients with congenital cataract operated on in adolescence or later; also its development in cases of prolonged blepharospasm which continue for many weeks or months in infancy or early childhood, and finally the meridional amblyopia of highly astigmatic eyes, first elucidated by G. Martin,[30] in which the meridian of greatest astigmatic diffusion is amblyopic.

That amblyopia ex anopsia is a true loss of vision and not a failure of its development is emphasized by Worth,[1] who cited numerous cases in which the central vision of the fixing eye deteriorated greatly in children under the age of 3 or 4 years, because its atropinization was accidentally so overprolonged that the primarily deviating eye became the fixing eye. He cited other cases in which after improvement of vision of the amblyopic deviating eye secured by occlusion or atropinization of the fixing eye, there was some relapse into amblyopia because the fixing eye was prematurely allowed an advantage. Luedde [27] reported similar experiences in six cases.

Improvement of vision of the amblyopic eye, by occlusion or atropinization of the fixing eye, was secured by Worth in the great majority of cases in which he was able to begin treatment early enough and to continue it long enough. The highest proportion of restoration of vision to normal was in the patients who received very early treatment. Similar evidence is given by other authors, especially most recently by Sattler,[31] who in 1927 reported marked improvement of vision, averaging an increase of twenty-nine times the original acuity in eighty cases in which the amblyopic eye had not lost central fixation. Most of these children were between the ages of 5 and 7 years, and the procedure used was occlusion of the fixing eye for a period of from one to twelve months. He also noted the more rapid improvement in the cases of most recent onset. In eyes with eccentric fixation, in some there was a restoration of central fixation followed by further improvement of vision, while in others eccentric fixation remained and there was some improvement of the eccentric acuity, though not to exceed 0.1 in any case, and this even with normal correspondence. Sattler [31] also observed a relapse into amblyopia of many eyes that had obtained good vision.

Even stronger evidence of the existence of amplyopia ex anopsia, if more were needed, is afforded by reports of marked improvement of vision of the amblyopic eye after loss of the fixing eye, or loss of its useful vision. Some of these patients were adults, and in some the amblyopic eye had not even central fixation. In three cases at least, vision improved to normal. One or more cases of this kind are reported by each of the following authors: Bielschowsky,[16] Herrenheiser,[32] Klein,[33] Heine,[34] Panas,[35] Schaaf,[36] Luedde,[27] Harris [37] and Johnson.[38] Johnson's two patients were both adults, who recovered normal vision in

fifteen days, one having previously had vision only of counting fingers in the amblyopic eye. In most of the cases, however, recovery was not complete and required months or years.

RETINAL INCONGRUITY

Retinal incongruity, or abnormal correspondence, is the third important disturbance of the visual functions in squint which claims attention. This condition is an abnormality of the sensoric relations of the two retinas which gives rise to disturbances of relative localization. The significance of these anomalies and the methods of examination by which they may be studied will be described in some detail, since there seems to be as yet practically no reference to this subject in any of the available literature in the English language. Two varieties of abnormal cor-

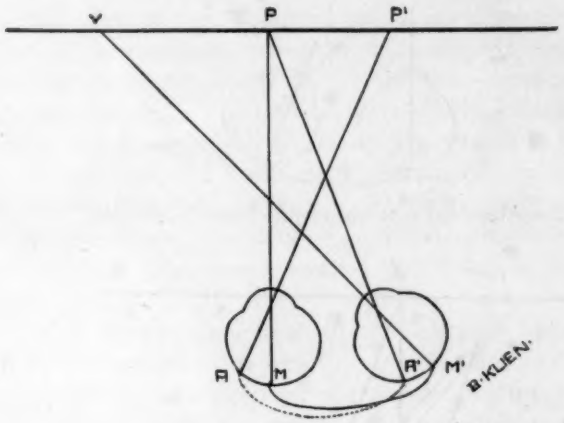

Fig. 1.—Normal correspondence.

respondence are to be distinguished: (1) the harmonious type, in which the angle of squint and the angle of anomaly are equal, and (2) the unharmonious type, in which the angle of squint and the angle of anomaly are not equal. The harmonious type is said to be relatively rare.

The conditions of relative localization in squint with normal correspondence are shown graphically in figure 1. The eccentric retinal point A^1 in the squinting eye receives the image of the point P fixed by the fixing eye. Since the two maculae M and M^1 are corresponding points, the point A^1 has the same visual direction * as A, hence localizes P at P.[1]

* The expression "visual direction" is used in a special technical sense, as a translation of the German "Sehrichtung" meaning direction of localization, and is not the same as visual line, line of fixation, optic axis, etc.

The localization in the harmonious type of abnormal correspondence is shown in figure 2. In this case, the two maculae are not corresponding points, but the point A^1 in the squinting eye, which receives the image of the point P, corresponds to the macula, M, of the fixing eye, i.e., has the same visual direction; so it localizes P in its true place, and there is no diplopia. The angle of anomaly and the angle of squint are thus equal.

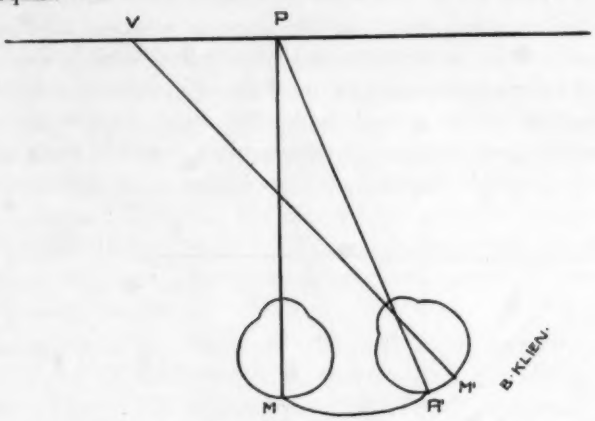

Fig. 2.—Abnormal correspondence, harmonious type.

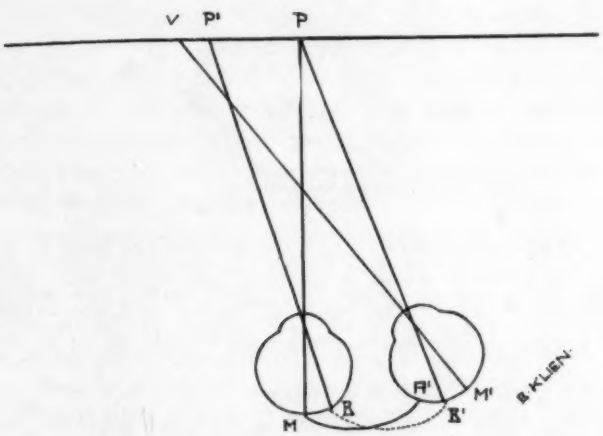

Fig. 3.—Abnormal correspondence, unharmonious type.

Localization in the unharmonious type of abnormal correspondence is shown in figure 3. In this case the point A^1 in the squinting eye has the same visual direction as the macula, M, of the fixing eye. The point B^1 in the squinting eye, which receives the image of the point P, corresponds to the point B in the fixing eye, which is the same distance and direction from M as B^1 is from A^1. Hence, B^1 has the same visual

direction as B, and localizes P at P'.[1] The unharmonious type of abnormal correspondence may be regarded as having developed out of the harmonious type by a diminution in the size of the squint angle, which was formerly so large that A^1 received the image of P.

It is interesting to note that in cases of squint with normal correspondence and in those with abnormal correspondence of the unharmonious type, two areas of maximal suppression are present in the squinting eye, namely, its own macula and the eccentric area receiving the same image as the macula of the fixing eye; while in the case of harmonious abnormal correspondence, there is an area of suppression in each eye, which is in each case the eccentric area that receives the same image as the macula of the other eye.

MEASUREMENT OF THE SQUINT ANGLE

Certain methods of examining the sensoric relations of the retinas require for their correct interpretation an accurate measurement of the squint angle, which is also necessary for some other purposes. Hence, at this point it may be well to consider the various methods of measuring the squint angle, which may be classified according to six different principles, as follows: (1) linear measurement, (2) methods requiring the use of the corneal reflex, (3) prism measurement of the screen deviation, (4) measurement of the blind spot displacement on the stereocampimeter, (5) the after-image method and (6) the method by double images.

The first three of these methods are essentially objective, the remaining three subjective. The most commonly used methods are those of the second group, which depend on the corneal reflex.

The first method is the simple linear measurement in millimeters of deviation of the temporal margin or some other fixed point of the cornea, using the strabismometer, or marking the relative positions on the lower lid. It is seldom used at the present time.

Methods of the second group, which use the position of the corneal reflex as a basis, depend on accurate location by the examiner, of the corneal reflex in the center of the cornea, the center of the pupil or successively in the same relative position on the cornea of the fixing eye and the squinting eye. Variations of 1 or 2 mm. in judging the exact position of the reflex can introduce errors of 5 or 10 degrees, or more, in the measurement of the squint angle. The keratoscopic arc of DeWecker and Masselon [40] adds considerable accuracy to these measurements. This arc has a central notched white disk for fixation and two lateral white disks each 70 degrees from the center. When the images of the two lateral disks are equidistant from the limbus on each side, then the central disk is known to be in the center of the cornea. One is restricted, at any rate, to the objective methods of measuring the squint

angle in young children, so that often these methods with the corneal reflex are the only ones applicable. The commonest of these is by means of the perimeter. Worth objected to this method as too time consuming, also because it does not take account of the angle gamma (or kappa) which must be measured separately and added (algebraically); he said further that it cannot be used for young children. The force of these objections, however, seems questionable. Worth [1] described three other methods of this type in which the angle is measured by the tangent instead of the arc, namely, the deviometer, the Maddox tangent scale and the Priestly Smith tape (the Holzer [41] modification of the latter method by a second radial tape utilizes the chord instead of the tangent). The validity of these methods depends on two questionable assumptions: (1) the equality of the angle gamma in the two eyes and (2) comitancy, which although it is a property inherent by definition, is lacking to some extent in those few early cases which have developed from a true muscle paresis, and also in those cases in which the patients later in childhood have developed secondary muscle changes or have had operations on the muscles. In addition to these theoretical uncertainties is the practical difficulty of preventing the deviating eye from assuming the fixing rôle as the fixation object is moved in its direction along the tangent arm.

The third method, that of prism measurement of the screen deviation (cover test), has been accurately described and advocated by Duane.[42] It can be applied in all cases in which the squinting eye has the power of central fixation, and its accuracy is further enhanced in some cases by a supplementary subjective parallax test. The measure of the angle is the value of the weakest prism base out, less 2^{Δ}, expressed in degrees of arc, which just reverses or overcorrects the movement of the deviating eye behind the screen, or more practically, the movement of redress when this eye is uncovered. Duane found this method satisfactory in the measurement of squint angles as large as 40 or even 50 degrees, and stated that it is accurate to a fraction of 1 degree for angles of 20 degrees or less.

The method of measuring the squint angle by outlining the blind spot of the squinting eye by means of the stereocampimeter is described and advocated by Lloyd.[43] Theoretically, the method is simple, the measure of the angle being afforded by the difference in location on the chart, of the blind spot outlined first with the squinting eye fixing (the other eye being occluded) and second in its squinting position (the other eye then fixing). Obviously it matters not whether the blind spot is normal or abnormal in size or outlines, so long as the form and size in the two different positions are the same or sufficiently similar to show the difference in degrees of location of corresponding points of the outlines. Lloyd [43] admitted that the angle of deviation in alternating squint is difficult or impossible of measurement by this method, because

of the difficulty of maintaining the eye under examination in the squinting position. He stated that the stereocampimeter measurement usually gives a much smaller angle than the perimeter, and that 20 degrees by the former method is a very large angle.

Measurement of the squint angle by the after-image method will be described in connection with the measurement of the angle of anomaly by the same method in order to emphasize the important difference of conditions for the two tests.

In cases with normal correspondence, the squint angle can be measured by the angular separation on a tangent scale at a given distance, of double images elicited by colored glasses or by the Maddox rod. Naturally, this method will fail in cases in which the suppression of images in the squinting eye is so strong that diplopia cannot be elicited. When there is abnormal correspondence, the angle between the double images will naturally differ from the squint angle by the amount of the angle of anomaly.

METHODS OF DETERMINING SENSORIC RELATIONS

Investigation of the sensoric relations of the retina of the squinting eye to those of the fixing eye may be made by three methods: (1) the after-image test of Tschermak,[44] (2) the "Congruenz-apparat" of Tschermak [44] and (3) the comparison of the squint angle with the angle between the double images elicited by red and green glass filters. When abnormal correspondence exists, the angle of anomaly may be measured by a method utilizing the after-image produced in the squinting eye, to be described later, or by determining the difference between the angle of squint and the angle of diplopia. For the production of suitable after-images for these tests, it is essential to use a dark room and a long tubular lamp with a bright filament, resembling Tschermak's or Hering's lamp. An added refinement is an opaque band around the center of the lamp in order to produce a small dark interval in the center of the after-image. When the test is being made, a horizontal after-image is produced in the fixing eye, then a vertical after-image in the squinting eye, the other eye being fully occluded in each instance. Fixation must be central, and at a distance of about 1 meter. When there is normal correspondence, the two after-images are seen to form a symmetrical cross, as in figure 4, *a*. If there is abnormal correspondence, the vertical image crosses the horizontal to one side of its center, as in figure 4, *b*. The test may be difficult at first in some cases because of marked alternation of the images with only fleeting simultaneous perception.

When there is abnormal correspondence, the angle of anomaly may be measured by the after-image method in the following way:

With the lamp in the vertical position, the after-image is produced only in the squinting eye, as previously described. The squinting eye is then occluded, or screened, and the patient is seated in a dimly lighted room at a distance of 1

meter from a tangent scale on the wall (or some other distance, as for example 60 cm., with its proper tangent scale, may be used). While the zero point of the scale is fixed by the fixing eye, a slender rod (or pencil) held vertically is moved along the tangent scale by the examiner until it coincides with the after-image, as designated by the patient. The angle thus measured is the angle of anomaly. In order to make this measurement, it is necessary for the squinting eye to localize its macular image in space in relation to the eccentric retinal area in the other eye to which it corresponds. Hence, the projection of the macular image along the visual axis of the squinting eye must be prevented by the occlusion or screening of the latter. This mechanism is graphically shown in figure 5. In order to measure the squint angle by the position of the after-image on the tangent scale, it is necessary to secure the projection of the macular after-image in the squinting eye along its visual axis, and to exclude the mechanism of relative localization utilized in measuring the angle of anomaly. For this purpose the squinting eye remains open, and the fixing eye is excluded by a screen with a small central aperture just large enough to permit macular fixation on the zero point of the tangent scale. This mechanism is graphically shown in figure 6.

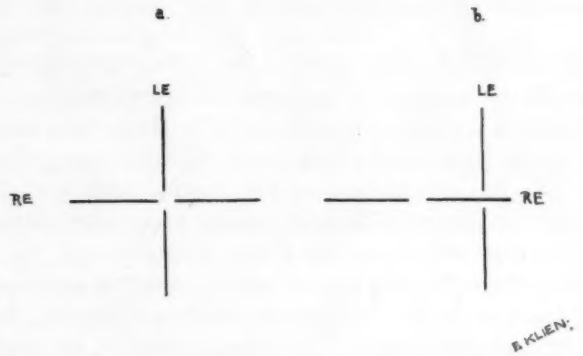

Fig. 4.—Tschermak's after-image test. Left convergent squint; *a*, normal correspondence; *b*, abnormal correspondence.

Tschermak's [44] "Congruenz-Apparat" for determining the type of retinal correspondence is even simpler for the patient than the after-image tests, and has the further advantage that it does not impose any artificial conditions on the formation of the images. The arrangement of the apparatus, in which a white cross lies midway between an upper green and a lower red slit, both of which are vertical and accurately alined with the vertical arms of the cross, is such that the red slit, below, is visible only to the right eye (being screened from the left eye by a blind), and the green slit, above, is visible only to the left eye (being screened from the right eye by a blind set at a similar angle but in the opposite direction). The patient observes this apparatus from a distance of 20 cm., fixing on the white cross, the angle of the blinds being arranged for this distance; it can easily be adapted to other distances if desired. The observations by this method, and their interpretation, are as follows:

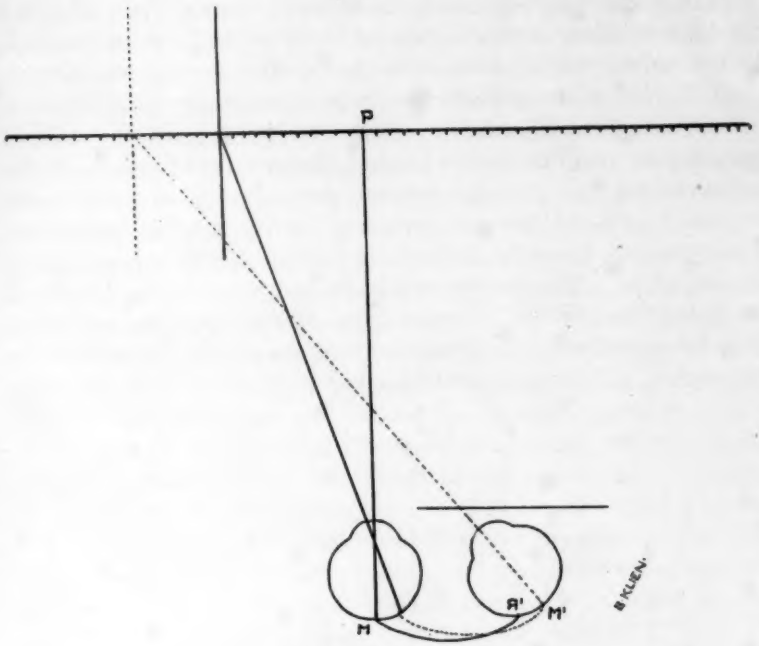

Fig. 5.—Angle of anomaly.

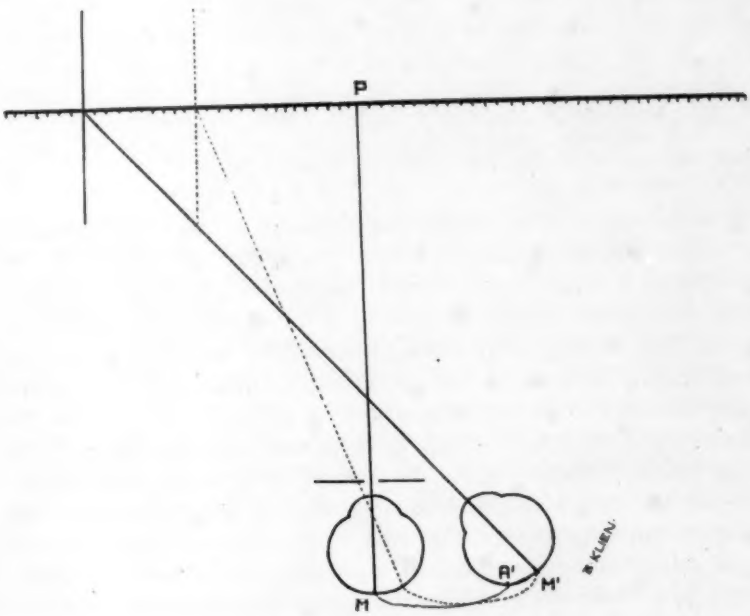

Fig. 6.—Angle of squint.

Assuming the squinting eye to be the left, the red slit (below) is seen to have the same vertical alinement as the vertical arm of the cross. If there is normal correspondence, the green slit is seen displaced to the left (fig. 7, *c*) by a distance which is the tangent of the squint angle; if there is abnormal correspondence, unharmonious type, with the angle of anomaly smaller than the angle of squint, the green slit is also seen displaced to the left, but a smaller distance than corresponds to the squint angle (fig. 7, *b*). If there is abnormal correspondence, harmonious type, the green slit is seen in perfect alinement just as by a nonsquinting subject (fig. 7, *a*). This test determines the sensoric value of the macula of the fixing eye and the eccentric area of the squinting eye which receives the same image, whereas the after-image test determines the sensoric values of the two maculae.

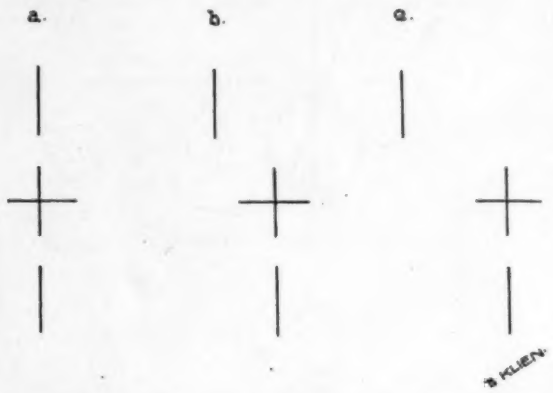

Fig. 7. — Tschermak's "Congruenz-Apparat" (left convergent squint). *a*, abnormal correspondence, harmonious type or no squint; *b*, abnormal correspondence, unharmonious type, and *c*, normal correspondence

The earliest observations concerning abnormal relative localization in squint were made by Johannes Müller [45] in 1826. He attributed the phenomenon to a congenitally eccentric position of the macula of the squinting eye. A few years later, von Graefe [12] was of the opinion that the incongruity is acquired in consequence of the squint. During the following decade, the trend of thought on this question was characterized by the development of the nativistic school of Hering,[46] who believed the correspondence of the two retinas to be present congenitally, and the empiristic school of Helmholtz,[47] who assumed it to be acquired in consequence of the desire for binocular vision. In accordance with the Helmholtz [47] empiristic theory, Parinaud,[48] in 1898, after describing the finding of abnormal correspondence in a certain group of cases of squint, stated his belief that perfect binocular vision with depth sense could be developed on the basis of abnormal correspondence. In this view,

however, he is almost alone, since Bielschowsky,[49] Adam,[50] Mügge,[51] Tschermak [44] and most other authors are agreed that the highest grade of binocular vision with depth sense can never develop on the basis of an abnormal correspondence. These observations must be considered as almost convincing evidence in favor of the nativistic theory of Hering.

At the end of the nineteenth century came the work of Tschermak,[44] who, having an alternating squint, elaborated by means of observations on himself methods of testing the retinal correspondence, at first with after-images, and later by means of the "Congruenz-apparat." He was the first to distinguish between the harmonious and the unharmonious types of anomaly. Then followed Bielschowsky,[49] Adam,[50] Schlodtmann,[52] Ohm [53] and Mügge,[51] with additional series of examinations of abnormal correspondence. The latter found this condition in 37.5 per cent of 120 cases of squint, and first emphasized its close relation to the true alternating type of squint, which has been confirmed recently by Braun. Mügge [51] further believes that abnormal correspondence develops before the sixth year of life. In support of this idea, he cited the cases of nine patients with alternating squint in whom the onset of the deviation was subsequent to the tenth year, in all of whom there was normal correspondence, in contradistinction to the fact that nearly all patients with alternating squint have abnormal correspondence.

The marked importance of the sensoric relations of the retinas with reference to the prognosis and therapy of squint has been shown recently by Braun,[54] of the Elschnig clinic. He examined a considerable number of cases, and described especially the observations in forty-six cases of convergent squint, which were exhaustively studied. In this series he found 60 per cent with abnormal correspondence, and divided all the cases into three groups on the basis of the sensoric relations, as follows: group A, those with constant normal correspondence; group B, the mixed group, in which at times normal and at other times abnormal correspondence prevails; group C, those with fixed or constant abnormal correspondence. He gave the prognosis concerning the development of binocular vision in these groups as follows: Full binocular vision, with depth perception, is usually possible in group A. In group B, the development of binocular vision is difficult, and does not usually succeed beyond the first stage, that of superimposition of images, or simultaneous perception without true fusion. In group C, binocular vision is never possible, and the squint angle must often be left partly uncorrected in order to avoid paradoxical diplopia. In some cases of this group the occurrence of paradoxical diplopia after surgical correction of the squint angle is obviated by the development of a strong suppression of the images in the squinting eye.

Abnormal correspondence, according to Braun,[54] Bielschowsky [49] and other authors, represents an adaptation of the sensory relations of

the retinas to the squinting position, which occurs in patients having an extremely marked congenital defect of the fusion faculty, as in these cases the normal correspondence seems to be very labile. Alternating squint is the usual type in this group.

CORRECTION OF DISTURBANCES OF VISUAL FUNCTIONS

The question of what one can do to correct these disturbances of the visual functions comprises a most important although too commonly neglected part of the orthoptic or nonsurgical therapy of squint, namely, the training of the fusion faculty and the prevention or cure of amblyopia ex anopsia. The significance of abnormal correspondence in this regard is that not only does it fail to offer in itself an objective for therapeutic efforts, but in the group with fixed abnormal correspondence there is not even a rudimentary fusion faculty to offer the possibility of training; at the same time, there is an alternating squint in most of these cases, so that amblyopia ex anopsia is the exception.

In training the fusion sense by means of the Worth amblyoscope, the necessity of beginning at an early age cannot be emphasized too strongly. When the child is 6 years old or older at the beginning of fusion training, the probability of success is much reduced, and the period of time required is greatly prolonged in those cases in which it is still possible to develop some degree of binocular vision. Worth [1] emphasized the fact that the relative ease or difficulty of eliciting diplopia by artificial means is only an indication of the depth of suppression of images in the squinting eye, and has no relation to the grade of binocular vision which it is possible to develop. In cases in which it is possible to determine the presence of a fixed abnormal correspondence, the effort to train the fusion sense by means of the amblyoscope either is without result or else produces a troublesome diplopia where none existed before. Since it is usually impossible to determine the nature of the retinal correspondence in children of 5 years or younger, the amblyoscope should be used in all cases in which a defect of fusion sense can be demonstrated. This is the more essential since it is probable, as Mügge [51] believed, that when a fixed abnormal correspondence occurs, it is acquired during the first five or six years of life, and the influence of fusion exercises with the amblyoscope during this period of conflict between abnormal and normal correspondence may suffice to prevent the development of the former and to establish the latter. Marked amblyopia in the squinting eye always prevents progress in fusion training, and Worth stated definitely that he never undertakes it in cases in which it is impossible to develop an acuity in the squinting eye which will enable it to see the 1 inch ivory ball at 6 meters. Hence, the use of the amblyoscope must often be preceded by measures designed to overcome the amblyopia of the squinting eye.

The two measures employed for this purpose are: (1) occlusion and (2) atropinization of the fixing eye. The method of occlusion of the fixing eye is used in all cases in which the squinting eye has an acuity less than 6/36 (according to Worth [1]), while for those of better acuity, the use of atropine in the fixing eye suffices. When occlusion is used it must be continuous, except for the occasional renewal of the occlusion pad, which need not be oftener than once a week. It must be continued until the acuity of the amblyopic eye improves enough for atropinization of the fixing eye to be substituted for it, or until it becomes evident that no improvement will occur. In cases in which central fixation has been lost, occlusion of the fixing eye for twelve months or longer may be required, as reported by Sattler,[31] who found that in most of the favorable cases there was first a recovery of central fixation, followed by gradual improvement of acuity, while in a few the eccentric fixation remained and there was some improvement of the eccentric acuity, up to a maximum of 0.1.

Control of the accommodative convergence excess in squint by the use of lenses correcting the hyperopia and astigmatism is too basic and well established to need comment. It may be noted, however, that the use of bifocals to curb further the accommodative convergence function at the near point was first advocated by Emerson [55] in 1905, later by Wendell Reber,[56] and more recently by Ryer.[57] Reber's [56] method was to add + 3 diopters for near vision to the full atropine correction at the age of 3 years or at the first refraction, and then to reduce the addition by 1 diopter each year thereafter. The wearing of prisms base out to stimulate fusion by bringing the two images close together was used extensively by Sattler [31] in 1927 (in 114 patients from the ages of 2 to 7 years), who began with prisms as high as 22 $^{\Delta}$ in some cases before each eye and gradually reduced the amount of prism until there was no longer any manifest squint without prism, and binocular vision was established. Hooren,[58] in 1920, and Landolt,[59] in 1924, also used prisms in the same way in a small number of cases, with similar results.

SUMMARY AND CONCLUSIONS

To summarize, the etiologic factors in squint are the primary factor, which is a deficiency of the fusion faculty, and the group of secondary factors, of which the most important is the convergence—accommodative excess of hyperopia.

The amblyopias of squint may be divided into three main groups: (1) those of organic origin, most of which are not true amblyopias in the strict sense of the word, (2) congenital amblyopia and (3) acquired amblyopia. There is definite and positive evidence of the existence of acquired amblyopia, and while it seems to be much more frequent than

the congenital type, there may be some basis for differences of opinion concerning its exact incidence.

The common methods of measuring the squint angle by means which utilize the corneal reflex have marked sources of error, and in suitable cases, prism measurement of the screen deviation, the method of double images or the after-image method should give more accurate results. To the methods of examination in most general use should now be added examinations of the relative localization or sensoric relations of the two eyes.

Abnormal correspondence is not congenital but acquired and seems to be an effort toward adaptation of the sensoric relations to the squinting position, which occurs only in cases with the most marked deficiency of the fusion faculty, in which the normal correspondence is especially labile. These conditions are most frequent in alternating squint. Attempts to train the fusion sense with the amblyoscope in patients with abnormal correspondence are usually futile or worse. In fixed or constant abnormal correspondence no binocular vision of any grade can develop after surgical correction of the squint angle, while in the inconstant type nothing more than simultaneous perception is possible. In all these cases, the angle of anomaly should be taken into account in planning a surgical correction, in order to avoid paradoxical diplopia.

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THE SYSTEMIC USE OF HYPERTONIC SOLUTIONS IN GLAUCOMA *

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The osmotic pressure of the blood can readily be increased by the intravenous injection of various crystalloidal or colloidal solutions (such as sodium chloride, dextrose or acacia) in the proper concentration. The effect of such a procedure on the pressure of the cerebrospinal fluid and the aqueous humor has been known by neurologists and ophthalmologists for a number of years. Matters of considerable importance in this connection, which have been carefully studied, are the similarity between these two fluids [1] and their probable dialysate nature, with the presumption that mechanical factors alone, and not secretory activity, enter into their formation.[2] Although we shall not touch on these topics, they satisfactorily explain the parallel responses of the aqueous and the cerebrospinal fluid to changes in the osmotic pressure of the blood.

In a recent paper on the treatment for glaucoma, Gifford [3] briefly reviewed the work which has been done on the systemic use of hypertonic solutions for the purpose of reducing abnormally high intra-ocular pressure. In the hands of several investigators employing different technics, a lowering of pressure could always be produced by this method.

Since, to the best of our knowledge, all the normal determinations had been done on animals, about a year ago Lambert and Silbert [4]

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reported the effect of intravenous injections of hypertonic solutions on the ocular tension of twenty-five normal men. An average drop of 40 per cent was found to take place in the ocular tension following the injection of 15 Gm. of sodium chloride solution, and a proportionate drop of 21 per cent with one-half that amount. Since that time, this method has been applied to a small series of cases on the service of Dr. Wolff. For the most part, the injections were given to prepare patients for operation.

The technic employed has been to inject from 200 to 300 cc. of a 5 per cent sodium chloride solution intravenously, by gravity, through a small bore needle. The use of a small needle automatically controls the speed of injection. The total dose of sodium chloride is thus seen to be 15 Gm. or less, which, in the absence of any contraindication such as nephritis, is entirely nontoxic. We do not believe that the addition of 300 cc. of fluid to the total volume of blood tends to counterbalance the osmotic action of the salt to any great extent. Higher concentrations of salt in less bulk are unsatisfactory, as they produce thrombosis of the vein used for injection.

Weight for weight dextrose has been found much less effective than sodium chloride. The reason for this becomes apparent when one considers that a 5 per cent solution of dextrose is isotonic to blood, as opposed to an 0.85 per cent solution of sodium chloride. The ideal solution would be colloidal in character, as it would have less tendency to permeate the capillary walls; it is interesting to note that Duke-Elder [5] succeeded in lowering the intra-ocular pressure in animals for a considerable length of time by the use of such solutions. They were not tried by us because of disastrous effects apparently from the use of acacia in saline infusions, reported during and immediately after the war.

REPORT OF CASES

CASE 1.—M. H., a man, aged 52, with bilateral acute glaucoma, had been treated with miotics for several weeks, but the tension could not be satisfactorily reduced. On admission, there was photophobia and slight pain in the eyes. Both eyes showed marked congestion, steamy corneas and shallow anterior chambers. The pupils were fixed, the right small, the left in semidilatation. The fundi could not be seen. Vision was reduced to the ability to see fingers at 4 feet in the right eye, and at a few inches in the left. The tension of both eyes was 62 mm. An injection of 200 cc. of 5 per cent saline solution was given, resulting in a reduction in the tension of both eyes to 45 mm. within one hour. The patient volunteered the information that his eyes felt "lighter" and better. After twelve hours, the tension had returned to 60 mm. No other form of treatment was used. Two more injections of saline solution were given, with a similar effect; after this, iridectomies were performed first on one eye, and then on the other. One injection, for some undetermined reason, produced a chill and a rise in temperature to

5. Duke-Elder, W. S.: The Pressure Equilibrium of the Eye, J. Physiol. **64**:78 (Oct.) 1927.

101 F., following which the patient experienced marked subjective improvement. (This is the only instance in which we encountered any febrile reaction.)

CASE 2.—H. A., a man, aged 60, with acute glaucoma of the left eye of one week's duration, was immediately put on miotic therapy. On his admission to the hospital, pain, photophobia and congestion of the left eye were noted. The cornea was hazy, and the anterior chamber was shallow. The pupil was dilated, and the fundus could not be seen. Vision was reduced to the ability to see finger movements at one foot, and the tension was 32 mm. The right eye was apparently normal. The patient was observed for five days, and as the tension could not be brought below 30 mm. it was decided to perform an iridectomy. Three hours before operation the tension of the left eye was 35 mm., and the eye was fairly painful. Three hundred cubic centimeters of 5 per cent saline was given intravenously. Just before the operation, the tension was 23 mm. and the patient stated that his eye felt much better. A definite improvement in the appearance of the cornea and the anterior chamber was noted by the surgeon at the time of operation.

CASE 3.—A. S., a woman, aged 64, with acute glaucoma of the left eye, had been seen previously, once in 1922 and once in 1924. It had been noted that the anterior chambers were shallow, but the tension of both eyes had been 16 mm. at each observation. At the onset of the present attack, the patient developed a tension of 65 mm. in the left eye, which was promptly reduced by miotics. Nine days later, after the treatment was relaxed, the patient had a second attack, with a tension of 70 mm. in the left eye. This was reduced to 22 mm. by miotics, and the patient was referred to the hospital. On admission, the eye was painful and congested, with a steamy cornea and a shallow anterior chamber. The vision was limited to the ability to see fingers at 8 feet, and the tension had returned to 60 mm. despite the administration of miotics. Two hundred cubic centimeters of 5 per cent saline solution was given intravenously, and two hours later the tension was 45 mm. An iridectomy was performed.

CASE 4.—M. P., a man, aged 60, with subacute glaucoma of the left eye, gave a nine months' history of periods of discomfort, but no acute attacks. Operation had been suggested several times but refused by the patient. When the patient was first seen, the tension of the left eye was 62 mm., and he was given an intensive course of miotics. On his admission to the hospital, the right eye was apparently normal. Vision was 15/15, and the tension was 15 mm. The left eye was slightly congested, the cornea was clear and the anterior chamber was somewhat shallow. The pupil was in middilatation in spite of the administration of miotics. The vision was 15/30, and the tension 30 mm. There was slight cupping of the disk. The nasal half of the field was contracted to between 20 and 30 degrees. It was decided to perform an iridectomy, and one and one-half hours before the operation 300 cc. of a 5 per cent saline solution was given intravenously. The tension of the left eye, taken just before operation, was 17 mm. The tension of the right eye was not taken.

CASE 5.—H. K., a man, aged 56, had bilateral secondary glaucoma, of long standing, as the ultimate result of syphilitic uveitis. When the patient was first seen in 1920, there were numerous opacities in the vitreous, a moderately high degree of myopia, incipient cataract in both eyes and a corneal opacity in the left eye. The Wassermann reaction was 4 +. An iritis developed in both eyes which responded favorably to local and antisyphilitic therapy. In 1928, a tension of 70 mm. developed in the right eye, with a hazy cornea, cloudy vitreous and much congestion. The same condition then developed in the left

eye. A number of attempts were undertaken to control the glaucoma and to restore vision, both lenses having become completely opaque, necessitating extraction. On the last admission, the left eye was quiet. The right eye was painful and congested; vision was 20/200, and tension 51 mm. The cornea was hazy, and there was an operative coloboma of the iris and aphakia. The fundus was indistinctly visible. There was absolutely no response to local medication, and the patient was receiving antisyphilitic therapy. Preliminary to a trephine operation, three hours preoperatively, the patient was given 300 cc. of 5 per cent saline solution intravenously. One-half hour after the injection, the patient stated that he could see better and that the eye felt less painful. Just before operation, the tension was 44 mm. and the eye was in a much more favorable condition for operation.

Case 6.—A. F., a woman, aged 40, with unilateral secondary glaucoma, gave a history of loss of vision and pain in the left eye for one week. On her admission to the hospital, the right eye was normal; vision was 20/20. The left eye was congested and tender to touch. The cornea was steamy, and the anterior chamber somewhat shallow. There were a moderate number of small deposits on Descemet's membrane. The pupil was in mid-dilatation and the fundus could not be seen. Vision was reduced to perception of light. The tension of the right eye was 19 mm., and the left 32 mm. On the night of her admission to the hospital, 200 cc. of a 5 per cent saline solution was administered intravenously. In one hour, the tension of the right eye was 16.5 mm. and of the left 23.5 mm. The cornea of the left eye was clearer, and the anterior chamber appeared deeper. The patient said that the eye was less sensitive to touch. The following morning, the tension of the right eye was 13 mm., and of the left 20 mm. The patient was then given a course of treatment with atropine, and investigations were made to determine the etiology of the condition.

Case 7.—E. B., a woman, aged 70, presented a case of secondary glaucoma of the right eye, following an attack of uveitis. The onset of the uveitis took place about six weeks before her admission to the hospital. For several days prior to admission, there had been severe pain and elevation of tension in the right eye. On admission, the tension was 44 mm. in the right eye and 12 mm. in the left. On examination, the right eye showed marked congestion, a steamy cornea and a shallow anterior chamber. The pupil was in semidilatation, and with the loupe a number of posterior synechiae could be made out. There were also a few scattered deposits of pigment on the anterior capsule. Vision was absent. The left eye was apparently normal. On the night of her admission, 200 cc. of 5 per cent saline solution was administered intravenously. The patient was in severe pain, but no sedatives were given. During the course of the injection, alleviation of pain started and continued until the patient went to sleep one-half hour later. The tension of the right eye dropped from 44 to 35 mm. within fifteen minutes after the injection. The following day, the tension being 35 mm., a second injection was given, which reduced it only to 32 mm. There was considerable symptomatic improvement, although the appearance of the eye remained about the same. The following day, a third injection reduced the tension to 27 mm.; then operative procedures were undertaken, first a paracentesis of the anterior chamber and afterward iridectomy.

Case 8.—S. R., a man, aged 85, with chronic simple glaucoma of the right eye, and absolute glaucoma of the left, had been treated with miotics in the dispensary. In spite of this, there were occasional rises of tension in the right eye, and gradually diminishing vision. The patient was sent to the hospital for observation and possibly for trephining of the right eye. On admission, the right eye showed a coloboma of the iris, where a previous iridectomy had been done. The

media were clear, and definite cupping of the disk could be seen. Vision was 20/70, and the tension 23 mm. The left eye was externally negative save for an atrophic iris. The pupil was small, and the fundus could not be seen. There was no vision or light perception, and the tension was 32 mm. On admission, 200 cc. of a 5 per cent saline solution was given intravenously to determine its effect. In forty minutes, the tension of the right eye had been reduced to 14 mm., and the left to 27 mm. After three hours, the tension of the right eye was 12 mm. and of the left 22 mm. The following morning, the tension of the right eye had returned to 20 mm., and that of the left to 27 mm. The patient was ultimately sent back to the dispensary for further treatment, having refused operation.

CASE 9.—B. G., a woman, aged 83, with absolute glaucoma of the right eye, and chronic simple glaucoma of the left, gave a history of having had her right eye operated on about ten years before. The vision of her left eye had been slowly diminishing despite continuous treatment with miotics. On examination, the right eye showed a shallow anterior chamber, and an atrophic iris with a wide superior coloboma. The lens was sclerosed, and the fundus could not be seen. Light perception was entirely absent, and the tension was 15 mm. The left eye also showed a shallow anterior chamber and an atrophic iris. The pupil was moderately dilated, and the lens partially sclerosed. The fundus details could just be made out, and they showed a marked pathologic excavation. Vision was reduced to the ability to see fingers at 2 feet. Tension was 35 mm. The reduction in vision was attributed to the glaucomatous, rather than the lenticular, changes. No operation was considered advisable, but 200 cc. of 5 per cent saline solution was administered intravenously to determine its effect. In forty minutes, the tension of the right eye was 13 mm., and that of the left 22 mm. The following day, the tension of the right eye was 12 mm., and that of the left 28 mm. A second injection of 300 cc. was given, and in forty-five minutes the tension of the right eye was 10 mm. and that of the left 18 mm. On the day after the second injection, the tension of the left eye was again 28 mm.

The Effects of the Administration of Hypertonic Solutions in Nine Cases of Glaucoma

Case	Type of Glaucoma	Initial Tension, Mm. Hg	Lowest Tension, Mm. Hg	Drop, Mm. Hg	Amount of 5% NaCl, Cc.
1	Acute	62	45.0	17.0	200
2	Acute	35	23.0	12.0	300
3	Acute	60	45.0	15.0	200
4	Subacute	30	17.0	13.0	300
5	Secondary	51	44.0	7.0	300
6	Secondary	32	23.5	8.5	200
7	Secondary	44	35.0	9.0	200
8	Chronic	32	22.0	10.0	200
9	Chronic	35	22.0	13.0	200
Average of ten normal cases				8.3	300
Average of ten normal cases				4.1	150

From the table, it can be seen that there was a definite drop in tension in every case of glaucoma, following the intravenous injection of 5 per cent saline solution. In the nine cases, the drop varied from 7 to 17 mm. of mercury. Normal observations from previous work [4] are included in the table for comparison and to show that the amount of drop is directly proportional to the amount of solution injected.

The factors controlling the amount of drop in the pathologic cases cannot be determined from such a small series. It is suggestive, however, that the greatest drop took place in two cases of acute glaucoma which had the highest initial tension of the series, despite the fact that these cases received 200 instead of 300 cc. of saline solution. In practically all the cases with originally increased tension, the drop was greater than in the controls with originally normal tension.

CONCLUSIONS

In view of the considerable amount of work which has been done on this subject, in addition to our own, there can be no doubt that the intravenous injection of a hypertonic solution will always produce a drop in ocular tension. This fact one might well have anticipated because of its physiologic rationale. The question now arises as to where the particular value of this method lies.

The drop in normal persons was found to be short-lived, rarely lasting more than two hours. In cases of glaucoma, it was more protracted, sometimes remaining effective for as long as twenty-four hours. It is not a curative agent, as in our experience it has never produced a permanent drop in tension unaided by other measures. Furthermore, there is no great necessity for using intravenous therapy for patients who respond well to miotics or the other usual forms of treatment, although it can do no harm.

Every one, however, is familiar with intractable cases of glaucoma on which the usual forms of medication have little or no effect, and it is primarily for these cases that we believe the use of hypertonic solutions intravenously might well be adopted. As an adjuvant to miotics and other medications, the hypertonic solutions appear to favor their absorption by lowering the tension. In this manner, cocaine is more readily absorbed at operation. The value of having high tensions reduced for operation is evident, if only for the diminished danger of hemorrhage and loss of vitreous. Finally, although the value of morphine in certain cases of glaucoma cannot be overestimated, pain and anxiety can be greatly relieved by hypertonic solutions, so that smaller doses of sedatives can be given to the advantage of the patient.

We reiterate that, as we are dealing with what may be called a physiologic rather than a pharmacologic phenomenon, no matter what its application may be, it possesses a gratifying certainty of action.

Notes and News

EDITED BY DR. GEORGE S. DERBY

SOCIETY NEWS

XIII International Congress of Ophthalmology, Amsterdam, Holland, Sept. 5-13, 1929.—The national committee in charge of the organization of the congress reports that to date (July 1, 1929) nearly 700 ophthalmologists have qualified for membership, and that 232 scientific communications, covering all branches of ophthalmology, have been received and will be presented during the meetings of the congress.

Additional ophthalmologists who desire to qualify for membership should send the subscription of 25 Dutch florins ($10 in American money) to the treasurer, Dr. H. M. Roelofsz, Director of the Incassobank, Heerengracht 531, Amsterdam, as promptly as possible.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Vision

STUDIES IN PERIMETRY: II. PRELIMINARY WORK ON A DIAGNOSTIC SCALE FOR THE COLOR FIELDS. C. E. FERREE, G. RAND and M. M. MONROE, Am. J. Ophth. **12**:269 (April) 1929.

For this second paper of a series, designed to make determinations for color similar to those made in the first paper for form, 126 of the same observers used as subjects in the first paper were tested. The range of variation found in the color fields was not in all respects encouraging. The reasons for this are cited. The results may be summarized as follows: 1. In general the emmetropes and hyperopes have the wider fields, the myopes and presbyopes the narrower, although this separation into two groups is not so distinct as that in the form fields. From largest to smallest, the groups rank as follows: emmetropes, hyperopes, myopes and presbyopes. 2. The range of variations is much larger than for the form field. 3. In general the widest range of variation for field was found for the myopic group and the narrowest for the emmetropic. The order from greatest to least is myopes, presbyopes, hyperopes and emmetropes. 4. The distribution is in general of the same type as that in the form field; that is, the results when plotted approximate roughly the normal or gaussian frequency curve.

Because of the greater range of variation of fields and the large amount of scatter among the cases with narrow fields, the use of color fields for diagnosis presents greater difficulties than the form field. The greater overlapping of the refraction groups adds to this difficulty. It can probably be safely concluded, however, that the field of an emmetrope or hyperope that has an average breadth of less than from 34 to 35 degrees for blue, 30 for red and 16 for green in eight principal meridians is suspicious. For a myope or presbyope the borderline probably is from about 32 to 34 degrees for blue, 28 degrees for red, and from 12 to 14 degrees for green. In case that area of field is taken as the index, the borderline for emmetropes and hyperopes may be taken as 50 sq. cm. for blue, 40 sq. cm. for red, and from 10 to 12 sq. cm. for green; for myopes and presbyopes, 40 sq. cm. for blue, 30 sq. cm. for red, and from 5 to 7 sq. cm. for green.

In the laboratory work, larger fields and a much narrower range of variation were obtained. A number of charts are shown.

W. S. REESE.

Refraction and Accommodation

COMPARATIVE ESTIMATIONS OF REFRACTION. H. G. A. GJESSING, Acta Ophth. **6**:22, 1928.

By means of the Snellen chart, the Thorner-Busch refractometer, skiascopy and Holth's kinescopy, Gjessing investigated the refractive errors in 460 persons. In all, 780 eyes were studied. He grouped the cases according to the amount of astigmatism noted. The eyes which showed a difference of less than ½ diopter between the principle meridians were considered as nonastigmatic and those which showed a difference of ¾ diopter or more were regarded as astigmatic. He states his belief that the Holth kinescope is superior in some respects to the refractometer and skiascopy, for it permits the correction of refractive errors

in eyes in which there is a significant obstruction in the media, in cases where the pupils are constricted and in malingerers. He finds, however, that the person examined must have a moderate amount of intelligence. The Thorner-Busch apparatus gave insufficiently high hyperopia or excessively high myopia in a number of cases. He concluded that in a majority of cases the results obtained with the four methods did not vary by more than ½ diopter. He believes that with practice, skiascopy is the most rapid method. With proficiency, the Snellen test gives almost identical results.

A. M. YUDKIN.

MYOPIA AS A CONSTITUTIONAL CHANGE. ANTON INCZE, Ztschr. f. Augenh. **67**:20, 1929.

After a careful analysis of the literature on myopia, Incze noted the following outstanding facts: 1. Myopia is inheritable. 2. The sclera is the site of alteration. 3. Close work plays little if any part in the development of myopia. His survey of 188 cases of myopia disclosed the presence of a certain asthenic tendency which he described as "asthenia universalis congenita stilleri." He does not believe that this observation is a coincidence, but regards the damage to the elastic tissue of the sclera in myopia as an expression of generalized weakness of similar mesenchyme-derived tissue throughout the body. The eyeball is elongated by virtue of a daily fluctuating hypertonia of the intra-ocular fluid on the weakened scleral tissue. Another characteristic of this asthenia is the altered endocrine condition — a lack of epinephrine — of the ocular tissue. He substantiates his view of a constitutional basis of myopia by offering the data of other observers. Incze mentions the possible inheritable tendency of this ocular condition, but does not give detailed remarks about its influence.

A. M. YUDKIN.

Experimental Pathology

EXPERIMENTAL TUBERCULOSIS OF THE SCLERA. EMANUELE PANICO, Ann. di ottal. e clin. ocul. **57**:225 (March) 1929.

The author reviews the few cases of tuberculosis of the sclera reported in the literature and some attempts to produce the condition experimentally. It is agreed rather generally that the sclera is especially resistant to infection by the tubercle bacillus. The author inoculated rabbits with dead and living tubercle bacilli subconjunctivally and by injection into the sclera. Inoculations by both of these methods produced a scleral nodule with involvement of the conjunctiva. Superficial ulceration developed with evacuation of a small amount of caseous material which was followed by healing with scar formation. Perforation of the sclera never occurred. The cornea showed edema near the lesion. There was some hyperemia of the iris, and in a few cases small nodules appeared in the iris. Tubercle bacilli were found in sections of all the lesions. A bibliography and photomicrographs are included.

S. GIFFORD.

Lids

TREATMENT FOR SPONTANEOUS ENTROPION OF THE LOWER LID: PATHOGENESIS. A. TERSON, Ann. d'ocul. **166**:89 (Feb.) 1929.

The term spontaneous is chosen as being the most appropriate for the type of entropion described. Other names applied are senile, acute, temporary and spasmodic. The various curative methods, operative and otherwise, are briefly described before the one advocated.

The description of the method selected was presented before the Belgian Ophthalmological Society on Nov. 27, 1927, by Professor Weekers. He advised the subcutaneous injection of 80 per cent alcohol into the affected lid. Terson, after cleansing the lid with iodine and alcohol, anesthetized a zone, horizontal to, and about 3 mm. from, the margin of the lid, with 2 per cent procaine. A few minutes later he injected a little less than 1 cc. of 80 per cent alcohol. Collodion and a light dressing were applied. The same night, disagreeable sensations were felt. The next day there was edema of the inferior lid with the ciliary border in good position. A small crust where the needle had been inserted remained for four or five days, then disappeared without any cicatrix. The author advises against injection of the alcohol at the junction of the middle and inner quarters of the lid, so as not to involve the area just beneath the inferior lacrimal point. Other chemicals suggested for use instead of 80 per cent alcohol were sodium salicylate and double chlorhydrate of quinine and 5 per cent urea.

As to pathogenesis, entropion arises on a normal ciliary border while the anterior portion of the lid, skin and peripheral muscles has lost its tone. Other factors — enophthalmos and subcutaneous edema — accentuate the condition.

H. SCARLETT.

Lacrimal Apparatus

PHYSIOLOGIC PROBLEMS OF THE HUMAN LACRIMAL PASSAGEWAY. T. FRIEBERG, Ztschr. f. Augenh. **67**:1, 1929.

The author criticizes various experimental procedures and the deductions which several investigators have made from their observations concerning the mechanism of the lacrimal apparatus. He does not agree with Rosengren, who assumes that the sac plays no part in the expulsion of fluid into the nose. He believes that the lacrimal apparatus functions as a suction and pressure pump, and during a short quick closure of the palpebral fissure, the process is more effective than in a complete spasmodic contracture of the lids. A negative pressure is produced in the lumen of the canaliculus and lacrimal sac by the action of the orbicularis muscle and surrounding tissue, and the fluid is aspirated slowly from the lacus lacrimalis. In default of lacrimal fluid in the lacrimal system, air is aspirated from the upper and lower lids of the passageway, and capillary surface tension is avoided. In the slowly closing movement of the lids the lumen of the canaliculus and sac is compressed and its content is forced to the nasal opening. The sphincter action of the punctum assists in the procedure. The author notes that the apparatus functions best when the punctum is close to the eyeball. He advises that the punctum be opened by a scissors incision rather than with a knife if the passageway is occluded, and if the canaliculus must be slit, the incision should be made on the conjunctival side of the lid.

A. M. YUDKIN.

Conjunctiva

TREATMENT WITH DIACETYLLANINE AND ALBUMATE OF SILVER FOR CONJUNCTIVITIS. H. BIRNBAUM, Ann. d'ocul. **66**:131 (Feb.) 1929.

A preparation of colloidal silver and tannin, or to be more exact, a combination of diacetyllanine and albumate of silver was first used by Sibert and Henryk Kohn of Breslau in the treatment of patients with conjunctivitis. It is used in a 5 per cent solution, one drop three or four times a day, in all forms of con-

junctivitis, acute or chronic. It causes an immediate constriction of the vessels and a striking diminution of secretion. In acute conjunctivitis this effect remains for several hours, and when secretion recommences it is quickly controlled by a fresh instillation. This drug causes the conjunctiva rapidly to return to normal. It is also recommended in conjunctivitis associated with rheumatism.

The most marked success has been attained in chronic conjunctivitis of all sorts and origins, especially the types that have endured for months or even years. In trachoma the treatment is indicated in two stages: first, in the period of infection and secretion, when it renders the lids sufficiently dry to treat properly or to operate; second, in the final cicatricial stage. 1. The preparation of diacetyllanine and albumate of silver infiltrates the tissues without precipitating albumin. 2. It is not an irritant, but an astringent. 3. It renders the tissues less favorable for the propagation of micro-organisms and is at the same time a disinfectant. As to its action on micro-organisms, a comparison is made between it and silver protein. To prevent a cultural growth 1:400 diacetyllanine and albumate of silver solution is employed, while 1:100 of silver protein is necessary. This is explained on the basis of the colloidal nature of the first preparation, which is poor in free ions of silver, and permits a quick and deep penetration of the tissues by the fluid. Those silver preparations which are rich in free silver ions act more superficially and do not penetrate the tissues so deeply.

H. SCARLETT.

Cornea and Sclera

DELAYED CORNEAL ULCERATION FROM MUSTARD GAS. R. FOSTER MOORE and FRANK HECKFORD, Brit. M. J. **1**:497 (March 16) 1929.

Two cases are reported because of their clinical interest and their importance from the point of view of war pensions. Case 1 was that of a physician who was burned by mustard gas in 1917. Corneal nebulae from ulceration of the left cornea, appearing in 1922, entitled him to a pension. Eleven years later, in 1928, the right cornea became spontaneously ulcerated, involving one third of the cornea. From the first, it was deep and crater-like, extending to Descemet's membrane. Gray lines of infiltration radiated from its border, a small area only was stained with fluorescein. The ocular conjunctiva presented a characteristic appearance. The symptoms were mild, and after five months the progress of the ulcer was arrested. At the end of seven months, repair is not complete. Case 2 was that of a healthy man aged 38 who received mustard gas burns of the body and eyes in 1917. He had no particular trouble until July, 1928, when small deep ulcers developed in both corneas and slowly extended; in November, 1928, a deep gutter-like excavation of corneal tissue was present in both eyes. The edges had a rolled appearance, looked sodden and stained with fluorescein. The cornea above showed linear infiltration and two small punched-out ulcers. The conjunctiva at one side was opaque and marbled in appearance.

The authors mention the general resemblance of this condition to Mooren's ulcer, the long period involved, roughly ten years, between the cause and actual development, the indolence and the characteristics and slow healing of the ulcers. They state the belief that the gas damaged the cornea and caused scarring which impaired its nutrition and led to ultimate ulceration. The treatment consists in curetting any staining area, careful cauterization and, possibly, sewing the lids together.

A. KNAPP.

TREATMENT WITH ULTRAVIOLET LIGHT FOR CORNEAL DISEASES. H. GASTEIGER, Klin. Monatsbl. f. Augenh. **82**:344 (March) 1929.

The treatment with ultraviolet rays, begun by Hertel in 1907, was temporarily abandoned in favor of ethylhydrocuprein, but was given more consideration by Birsch-Hirschfeld again, after the action of the drug failed to come up to expectations.

Gasteiger reports the results obtained with this treatment at the Innsbruck clinic during the last five years. For details of the technic he refers to Birsch-Hirschfeld and Hoffmann's book, "Light Treatment in Ophthalmology." His report comprises seventy-five cases of ulcus serpens, thirty-two of which were very serious, involving a considerable portion of the cornea and showing high hypopyon and chemosis. *Pneumococcus* was found in fifty-eight cases, eleven of them being less severe conditions; *Diplococcus* Morax-Axenfeld in sixteen; *Bacillus coli* and white *Staphylococcus* in one case. Sixty-four eyes healed without perforation. Two of the eleven perforations were caused by conditions not related to either the disease or the treatment. Enucleation was required in two cases: in one for corneal staphyloma, in the other when secondary glaucoma had developed six months after treatment was discontinued. Vision was retained in sixty-nine eyes. Forty-eight patients had only atropia and hot applications, besides the light treatment, others received ethylhydrocuprein or zinc sulphate in addition. In a few cases perforation of the anterior chamber was resorted to. In others, thermocautery or Wessely's steam cautery was applied.

Gasteiger compares these results with those obtained in sixty-nine patients treated during the five years preceding the time when ultraviolet light was introduced: nine enucleations had to be made. He found that the ultraviolet treatment reduced the losses from 20 to 8 per cent. In accordance with Hertel, he states that the corneal scars are generally fainter than those caused by thermocautery and that the resulting vision is better.

Ultraviolet treatment proved its merit, furthermore, in twenty-two of twenty-six cases of corneal herpes; some of these patients were treated besides with a bismuth preparation or Balsam of Peru salve, or were curetted. The use of ultraviolet rays is suggested in filiform keratitis. No results were obtained in interstitial or deep-seated keratitides. Corneal wounds with infections, present or suspected, yielded prompt results.

K. L. STOLL.

Lens

CATARACTA ZONULARIS PULVURULENTA. E. M. PETERDY, Arch. di ottal. **1**:35 (Jan.) 1929.

Under this name, the author describes the condition found in a mother and two sisters; the details of the condition were revealed by slit-lamp examination. All showed bilateral punctiform lenticular opacities, arranged in concentric layers about the embryonal nucleus, with clear spaces between the layers. In the daughters the opacities involved the cortex almost to the capsule, while in the mother, aged 41, the cortex and senile nucleus were relatively clear, except for the disk of opacities of the embryonal nucleus. The central opacities were dustlike, while the more peripheral opacities were larger white globules. Vision varied from 2/10 to 6/10. Among the six siblings, two others, as well as the grandfather and a maternal uncle, were probably similarly affected. Related cases of this combination of embryonal nuclear cataract with zonular opacities described by Vogt, Poos and the reviewer are mentioned.

S. R. GIFFORD.

Glaucoma

TREATMENT OF GLAUCOMA BY ADRENALIN AND HISTAMIN. W. S. DUKE-ELDER and FRANK W. LAW, Brit. M. J. **1**:590 (March 30) 1929.

Epinephrine and histamine are first defined and their action is described as at present generally interpreted. Their action on the eye is then described according to the data of various experimenters. The authors regard Hamburger's explanation of their action in glaucoma as unconvincing and difficult to follow.

Two epinephrine substitutes, laevo-glaucosan and amino-glaucosan, were employed just as Hamburger recommended in seven cases of chronic glaucoma, in two cases of secondary glaucoma and in six cases of acute glaucoma, with the following conclusions: 1. So far as can be seen, neither of the two drugs has any prolonged effect in lowering the intra-ocular tension. Their action would seem to be temporary and adjuvant to other treatment, postponing, perhaps, but not without any special reason replacing, operation. The action of the drugs is neither uniform nor dramatic.

2. In chronic primary glaucoma, laevo-glaucosan cannot be depended on to lower the tension; it does so in some cases, but completely fails in others. It appears, however, that in connection with eserine a good effect can be produced in a case where eserine alone has remained ineffective.

3. The dilatation of the pupil caused by laevo-glaucosan may be valuable from a diagnostic point of view, since, as far as the observations go, the dilatation is not accompanied by raised tension.

4. In secondary glaucoma, laevo-glaucosan will succeed in rupturing synechiae which are not affected by other mydriatics. In cases of iritis with raised tension, it may be effective as a preliminary measure when hesitation is felt in giving atropine at once, or when atropine has failed to relieve the condition. Owing to the accompanying anemia, however, its repeated use appears to be inadvisable.

5. Amino-glaucosan is an extremely potent miotic. It cannot be depended on to contract the pupil and lower the tension in every case of acute glaucoma, but it would appear on occasion to be a useful adjunct to eserine. In cases in which the pupil remains dilated and the tension is raised after the administration of amino-glaucosan, this preliminary treatment has undoubtedly made eserine effective subsequently in bringing about a contraction of the pupil and a lowering of the tension. No case has been met with in which the drug has caused a rise of tension (as has been reported), but in several cases, although given every legitimate opportunity, it has had no hypotensive effect.

6. Both drugs are accompanied by a severe reaction and pain, in some cases moderate, in others involving considerable distress. The occurrence of a hypopyon ulcer without other obvious cause, after the use of amino-glaucosan, suggests that the severity of the reaction on the superficial parts of the eye is not without danger to the cornea, especially in cases in which infection is known to be present. A complete bibliography is appended.

A. KNAPP.

Retina; Optic Nerve

TRAUMATIC DETACHMENTS OF THE RETINA AND OPERATIVE TREATMENT. H. DUBOIS, Ann. d'ocul. **166**:81 (Feb.) 1929.

The prognosis of traumatic detachment of the retina is generally favorable, which may be true in cases due to hemorrhage or edema. There are certain cases, however, in which it is not so favorable, such as when a rupture permits

the vitreous to pass behind the retina. This may happen: (1) from tearing of the retina away from the ora serrata by a violent contusion or (2) from a penetrating wound producing a contracting band. In these, there is a much graver prognosis. The more passive the detachment of the retina, the more chance of operative success. By passive is meant the appearance of the liquid behind the retina by passage through the rupture. If the tear is closed, the detachment is healed, but if the detachment is active in which the fluid passes behind the retina secondary to traction in the vitreous, and if the vitreous traction is greater than the cicatricial traction produced by operation, the chances for a cure are fewer. The operative procedure employed for detachment of the retina is a puncture by means of the thermocautery through the sclera and choroid into the subretinal fluid, from about 13 to 19 mm. back from the cornea, depending on the location of the detachment.

The author concludes that traumatic detachments with a rupture at the ora serrata and without alteration of the vitreous usually heal rapidly and completely. Traumatic detachments resulting from a cicatricial band, due to the presence of small foreign bodies, also permit of a favorable reapplication. When the retinal tear is due to vitreous traction, following the entrance of a large intra-ocular foreign body or a penetrating instrument, the chances of a cure are problematic.

H. SCARLETT.

TREATMENT IN OPTIC NEURITIS BY REMOVAL OF THE MIDDLE TURBINATE AND OPENING OF THE SPHENOIDAL SINUS. E. ESCAT and H. FRENKEL, Arch. d'opht. **45**:353 (June) 1928.

In reviewing the work of de Lapersonne and Truc, and of Valude and Frenkel, reported twenty years ago, the authors made the following statement in regard to the ocular complications of sinusitis: "In regard to the optic nerve they are characterized by hyperaemia of the disc, circulatory disturbances; unilateral optic neuritis, transient or of long duration, more common in disease of the sphenoid than in disease of the ethmoid and maxillary antrum; thrombosis of the central retinal vein, retrobulbar neuritis with sudden loss of vision. All these conditions can result in optic nerve atrophy. Canalicular retrobulbar neuritis and optic neuritis of nasal origin were the reason for Onodi's extensive research. His anatomical preparations show the close relationship of the optic nerve and chiasm with the posterior ethmoidal cells and sphenoid sinus, the extreme thinness of the bony wall between the optic nerve and the accessory sinuses of the nose, the course of the optic nerve in relation to them, all explaining very well the effect of disease of the sinus upon the optic nerve. Onodi describes thirty-five different types of relationships between the optic nerve and the ethmoid and sphenoid sinuses."

For twenty-five years one of the authors has referred to the other the majority of his cases of unilateral optic neuritis in which there was no evidence of syphilis, chronic interstitial nephritis, diabetes, multiple sclerosis or dental infection. The results of the examination have invariably been negative, and the authors have never had to intervene on account of suppuration in the sphenoidal sinus.

Since the war many reports have appeared of patients with optic neuritis cured by opening the sphenoid sinus, sometimes because of suppurative infection, but more often in the absence of any clinical evidence of suppuration. In Florent's thesis, Rollet differentiates between three types: (1) the typical form with central scotoma; (2) an atypical form described by Weil as characterized by central

scotoma with a greater contraction of the field of vision or papillitis with hyperemia of the disk, a surrounding halo and a slight modification in the caliber of the veins, and (3) the atypical form of Ronne with central and hemianopic scotomas, and evidence of papillitis and of chiasmal involvement. He insists on the differential diagnosis between the types of toxic neuritis due to alcohol, syphilis and multiple sclerosis, respectively. In agreement with Gargnon, he advises nasal and, more rarely, sphenoidal treatment.

Carrette has been able to assemble the cases observed into two groups, the first revealing a posterior sinusitis, and the second, a normal mucous membrane or a mild inflammation. His conclusions are: "Although in the course of an optic neuritis, suppuration of the posterior sinuses fully justifies nasal intervention, the relation of mild and latent infections of the same sinuses and the usefulness of opening them in these cases is not yet definitely established. Mild nasal infection seems relatively frequent in the acute forms of optic neuritis. Nevertheless, they are often cured spontaneously."

In the forms of slower development, with indefinite etiology, Bourgnet obtained five cures in five cases by intranasal intervention, and Baldenweck performed two successful operations in five cases.

In the cases in which the etiology is not known exactly, opening the posterior sinuses appears to be an empiric operation that only the progressive development of the neuritis can justify. In the old cured cases, the success of the operation is undoubted and is attributed to a reflex action favorably modifying the condition of the optic nerve. Experience shows that some nasal procedures are capable of modifying the circulation of the optic nerve, as, for example, the application of cocaine and epinephrine to the nasal mucous membrane. In reviewing the opinions which have been expressed, Carrette says: "Certain authors deny all relations between the intervention and the cure of the ocular accidents. Others think that sinus origin must be suspected in all cases of neuritis which do not show an acute or chronic intoxication, diabetes, syphilis, multiple sclerosis or heredity."

The authors then report seven cases in which benefit was obtained by opening the sphenoidal sinus. In their cases it was not so much a question of retrobulbar neuritis as of papillitis sometimes with beginning atrophy of the nerve.

In the four fully reported cases, the operation consisted of opening the sphenoidal sinus after removing the middle turbinate.

The following theories have been advanced to explain the resolution of the optc neiuritis: (1) relief of congestion by bleeding from the neighboring opening; this cannot be supported because little hemorrhage followed the operation in the four cases; (2) vasomotor reflex action; it is the only explanation according to the majority of authors, and there is certainly a great deal of truth in it, and (3) the suggestion of Escat that it may be due to the escape of lymph which always results when any solution of continuity of tissues takes place and which escapes attention because of its slowness and lack of color. The connection between the lymphatics of the pituitary and the subarachnoid spaces have been demonstrated by Axel Key and Retzies and confirmed by Andre. They explain well the beneficial effect on the optic nerve.

S. B. MARLOW.

EDEMA OF THE OPTIC DISK AFTER SPINAL PUNCTURE. LEVY-VALENSI, J. LAMACHE and DUBAR, Médecine **10**:122 (Feb.) 1929; abstr., J. A. M. A. **92**:1639 (May 11) 1929.

Two cases of edema of the optic disk with retinal arterial hypertension are reported which occurred several days after a spinal puncture. The condition was

afebrile and was associated with bradycardia, vomiting and cephalalgia in both patients, and with diplopia in the first patient. The patient was cured in a short time, and the condition did not leave any traces. Because symptoms of an instability of the sympathetic nervous system were observed in both patients before spinal puncture was performed, the authors state their belief that the condition was of vasomotor origin.

W. ZENTMAYER.

Neurology

CHANGES OF THE IRIS IN TABES. KAMINSKAJA-PAWLOWA, Klin. Monatsbl. f. Augenh. **82**:340 (March) 1929.

Ptosis, anisocoria and rigidity of the pupils cause the "brilliant but dead expression" in the eyes of tabetic patients. This picture is rendered more conspicuous, in the writer's opinion, by actual changes of the iris tissue, e. g., atrophy of the trabeculae and diminution or obliteration of the crypts. The eyes of twenty-five tabetic patients, aged from 30 to 50, were examined by means of the Zeiss binocular microscope. Their condition comprised every stage of tabes. The lifeless expression of the eyes was evident in every case, and was especially marked in light-colored eyes. The trabeculae were thin, resembling white threads in some instances; the crypts were shrunken; the posterior pigment layer was atrophic, especially at the margin of the iris. In one patient, it was fenestrated. The pigment lay like dust on the surface of the iris or, occasionally, on the posterior surface of the cornea. Sector-shaped atrophies of the iris were also observed.

The changes in the relief of the iris are of a physiologic or pathologic nature. The iris is physiologically smoother in miosis owing to the tightening of the tissue or in old age due to senile atrophy. Pathologic atrophy may be caused by protracted inflammatory processes which may also cause synechiae or deposits of pigment on the capsule of the lens or increased intra-ocular tension. Atrophy, furthermore, may be of neurotrophic origin, and this in the author's opinion is the cause of atrophy of the iris in the cases here reported, none of which showed evidences of inflammation, glaucoma or symptoms of a senile type. The actual cause of this atrophy is thought to be a disease of the sympathetic nerve, analogous to other diseases of the sympathetic system in the iris, e. g., heterochromia in encephalitis or changes of the color of the iris in syringomyelia. Changes of the autonomous ganglions will produce the sector-shaped atrophies; changes of the local nerves will cause the changes of the stroma. Inhibitions or irritations of the sympathetic system may be of consequence in decreasing the nutrition of the iris. This atrophy of the iris in tabes, which is neurogenic, is considered characteristic and thought to form a typical symptom. It occurs as frequently as the Argyll Robertson pupil and more frequently than optic atrophy; hence it is of considerable diagnostic value.

K. L. STOLL.

Ocular Muscles

ROTATION REFLEXES OF THE OCULAR MUSCLES. LORENTE DE NÓ, Acta oto-laryng. **12**:238, 1928.

Experiments on rabbits in which the tonus of the individual ocular muscles are recorded show the following reflexes on rotating the head. These reflexes change with alteration in position of the head in space. This paper deals exclusively with the changes occurring when the head is turned and the body is in the normal

erect posture. 1. Nystagmus: The usual changes are described. 2. Tonic reflexes due to the centrifugal force: This force in connection with gravity causes a tonic contraction of certain of the muscles of the eye. These changes are constant as long as the rapidity of rotation remains the same. 3. The third rotation reaction, "Dritte drehreaktion": This consists for the most part in a rapid contraction, but occasionally in a relaxation occurring at the beginning and at the end of rotation and lasting only a short time. It occurs only when rotation is begun and stopped quickly. The changes in this type of reaction and its causation are described. One illustration is given.

F. H. ADLER.

Orbit Accessory Sinuses

A CASE OF PULSATING EXOPHTHALMOS. P. SCOTTI, Ann. di ottal. e clin. ocul. **57**:248 (March) 1929.

A woman, aged 28, was struck in the submental region by a fragment of a revolver, which lodged in the nasopharyngeal wall near the sphenoidal sinus. From this location it later disappeared by erosion into the pharynx. Ten days after the injury, a souffle began, synchronous with the pulse; it was at times so loud as to prevent the patient from sleeping. Exophthalmos was noted shortly after this and the vision, which had been normal immediately after the injury, diminished to 7/10. The ciliary and retinal veins were markedly dilated and a venous pulse was visible. Fields showed a slight bitemporal contraction. Paresis of the sixth nerve was present. Eight months after the injury, the common carotid was ligated with marked immediate relief from the subjective symptoms. Six months after the operation, some of the symptoms returned but were not as severe as before. Vision remained 7/10 and the exophthalmos was slightly diminished. The condition is explained as due to a rupture of the carotid into the cavernous sinus, the injury being produced by a fragment of the sphenoid bone. Such cases are especially important from a medicolegal standpoint, and the prognosis should always be guarded.

S. GIFFORD.

General Disease

TRANSIENT OPTIC NEURITIS IN GRIP. K. SABATZKY, Klin. Monatsbl. f. Augenh. **82**:531 (March) 1929.

The author reports eight cases of optic neuritis observed during the last epidemic of grip which he attributes to this disease, after eliminating all other etiology. No neurologic symptoms were present. No medication was responsible. Complicating diseases of the nasal cavities or of the ears did not exist. All of these patients were seen within a period of four weeks. In some of them the eyes showed the first symptoms of the disease, while in others the eyes became affected during convalescence. None of them showed physical or psychic inhibitions of the central nervous system, signs of cranial pressure or meningeal irritation. The patients complained of indistinct or distorted vision or scintillations. The disk was edematous in every case, its margin indistinct in some of them, and slightly swollen in one case. The veins were strongly filled; the arteries were usually thin. The function of the eyes was otherwise normal, except that the vision was slightly reduced in some cases, and from one-third and to one-half in others. Fundus and vision were normal again after periods of from two to five days.

K. L. STOLL.

COMPRESSION OF THE THORAX FOLLOWED BY IMMEDIATE AND PERMANENT BLINDNESS. P. PICARD and P. A. DREYFUS, Arch. d'opht. **45**:383 (June) 1928.

The authors begin their report with a description of this condition. Such serious complications as amaurosis and lasting blindness are rare. Only a few cases have been reported. In 1906, Beal published the first report of a case. The patient did not become totally blind. Moser reported a second case during the same year. The right eye was totally blind with optic atrophy, but in the left eye the vision was 5/25.

In 1909, Beal reported the third case. The right eye became totally blind from optic atrophy; the left eye, however, was unaffected. Lang reported a case in which the central vision of the right eye was normal although there was a sector of atrophy of the optic disk with a corresponding scotoma. The left eye was blind.

Roenne's case showed no lesion when first seen, but the left eye became blind three days later, complete atrophy gradually ensuing. Stoewer's patient had narrow retinal arteries with engorged veins, linear hemorrhages and a grayish-white pallor of the retina of the right eye, which finally became blind.

LeRoux's case differed in that on ophthalmoscopic examination a week after the injury an edematous disk and engorged veins were discovered. Postneuritic atrophy finally occurred.

The authors' case is summarized briefly as follows: A man, aged 37, was crushed while lying face down during the operation of a large crane. He had a sensation of a rush of blood to his head and felt that his eyes "would burst out of his head." One or two minutes later, he could see nothing, not even light. He did not lose consciousness. On admission to the hospital he had the classic ecchymotic mask, which included the face and neck. He was clear mentally. There were several fractured ribs and an acromial dislocation of the clavicle. There were marked ecchymoses of the lids of both eyes, severe chemosis and subconjunctival hemorrhage and bilateral proptosis, more marked on the right. Motility was normal. The pupils were dilated and inactive. The cornea and the anterior segment were normal. The media were clear. The fundi, especially in regard to the disks and vessels, were normal except for an old pigmented choroidal lesion in the left eye. Vision was completely abolished; the tension was normal.

The subcutaneous ecchymoses disappeared in about two weeks; the subconjunctival hemorrhage lasted about seven weeks. The proptosis of the left eye subsided in six days, and of the right eye in eight days. In from twelve to fourteen days after the accident the disks began to pale.

Further study by x-rays revealed no bony lesion of the skull. The Wassermann reaction was negative. Examination of the vestibular apparatus indicated irritation of the right labyrinth. Spinal puncture resulted in negative Wassermann and colloidal gold reactions. The spinal fluid contained 13 cells. Results of a psychiatric examination were negative. The points of particular interest, according to the authors, are the fact that the patient never lost consciousness, the blindness that came on during the compression and the unusual duration of the exophthalmos.

The authors say that they cannot explain the case on the basis of orbital hemorrhage alone. They think it more likely due to hemorrhage into the optic nerve sheath posteriorly. All the symptoms can be explained in this way, especially since the reports of Elschnig, Gonin and Dupuy-Dutemps have shown that the condition can occur without any symptoms in the fundus.

S. B. MARLOW.

Tumors

RHABDOMYOMA OF THE EYELID. L. L. MAYER, Am. J. Ophth. **12**:293 (April) 1929.

The author refers briefly to the few cases on record and cites the case of a man, aged 66, who developed itching and burning of the right lower lid with epiphora. The lid started to swell and became larger. He was treated with a caustic for six months. When first seen there was a small, lumpy growth near the inner canthus. The lower lid was drawn down, exposing the conjunctiva, especially near the tumor at the inner canthus. There was marked dermatitis of the skin over the zygoma.

The patient was referred to the skin clinic, where a diagnosis of epithelioma basocellulare was made. The radiologic department advised against the use of radium. On Sept. 20, 1928, the tumor was excised and a diagnosis of rhabdomyoma was made microscopically.

W. S. REESE.

THE DIAGNOSIS, PROGNOSIS AND THERAPY OF ORBITAL TUMORS. STUELP, Ztschr. f. Augenh. **67**:41, 1929.

Stuelp describes in detail seventeen cases of intra-orbital tumors and the methods which were utilized for their diagnosis. He classifies the tumor masses found in the orbit into three groups: nonmalignant, malignant and pseudotumors. In the first group are (1) cysts: serous, oily, mucocele, echinococcus; (2) blood and lymph engorgements: hemangioma and lymphangioma, and (3) solid tumors: lipoma, fibroma, neurofibroma, chondroma and osteoma. The group of malignant tumors is comprised of (1) sarcoma and its related forms, and glioma and (2) carcinoma, cylindroma and mixed tumors of the lacrimal gland. The pseudo tumors are (1) syphilitic growths, (2) tuberculous masses and (3) leukemic tumors. The author's scheme of investigation is (1) determination of the personal and family history; (2) inspection of the orbital tissue and measurement of the exophthalmos with the exophthalmometer, and (3) palpation of the eyeball and its reductiveness. This procedure is best performed under scopolamine and morphine narcosis and temporary blepharotomy; (4) transillumination and roentgen examination; (5) detailed examination of the fundus with the ophthalmoscope and the slit-lamp, estimation of the visual acuity and peripheral field and the determination of the presence of diplopia; (6) careful general physical examination to rule out the pseudotumor group; it is necessary to make an analysis of the urine to determine the presence of melanin; also tuberculin and Wassermann tests are needed; (7) a chemical analysis of a diagnostic aspiration of fluid from the involved area and a microscopic examination of a diagnostic excision of the tumor mass. The prognosis of the case depends on the foregoing data.

A. M. YUDKIN.

Sympathetic Ophthalmia

POSSIBLE CURE IN SYMPATHETIC OPHTHALMIA WITHOUT ENUCLEATION OF THE TRAUMATIZED EYE. CALDERARO, Ann. di ottal. et clin. ocul. **57**: 194 (March) 1929.

While recognizing the beneficial effects which may be obtained by enucleating the traumatized eye in sympathetic ophthalmia, especially in the milder form of this disease, the author believes that in certain cases in which this eye still possesses vision it may be retained. He reports the case of a woman with definite sympathetic ophthalmia following an injury to the left eye which had resulted in a sub-

conjunctival scleral rupture with prolapse of the lens under the conjunctiva. No wound of the conjunctiva could be discovered. Vision was limited to the counting of fingers at a distance of 1 meter. The right eye showed marked iridocyclitis with vision reduced to the counting of fingers at a distance of 3 meters. The lens with prolapsed uveal tissue was excised, and some remaining uveal fragments were destroyed with the cautery. The patient was given atropine and potassium iodide, and in the course of three weeks the inflammation had quieted down in both eyes; vision was 1/10 in the injured eye and 1/2 in the other.

S. GIFFORD.

General

THE OCULAR SEQUELAE OF WAR GAS. J. A. PARLANGE, Arch. d'opht. **46**:87 (Feb.) 1929.

Little has been published in regard to the late effects of war gas on the eyes. The ocular sequelae can be divided into two classes: the undoubted cases and a second heterogeneous group including a variety of conditions. This second group includes cases of atrophy of the optic nerve, thrombosis of the central retinal vein, nystagmus, embolism of the central retinal artery, retrobulbar neuritis, glaucoma and acute spontaneous intra-ocular infection. The first group of cases the author subdivides into the cicatricial sequelae and what he designates as a conjunctivo-limbal syndrome.

The cicatricial cases have conjunctival and corneal scars due to secondary infection in areas of epithelial denudation resulting from the chemical action of the gas. In the conjunctivo-limbal syndrome, the blood vessels of the conjunctiva and limbus have become dilated and increased in number and volume to form, in some instances, varices. The limbus shows an opaque change and there is also some loss of sensation of the cornea near the changes in the limbus. Repeated attacks of conjunctivitis are common.

The author concludes from the observation of these cases and a few experiments on the eyes of rabbits that the vessels themselves have been damaged by the gas.

Treatment is subdivided into: (1) prevention of the attacks of conjunctivitis with drops and the removal of irritative factors, (2) treatment during the acute attacks and (3) control of the conjunctival hyperemia so far as possible with zinc sulphate and epinephrine.

S. B. MARLOW.

Society Transactions

EDITED BY DR. GEORGE S. DERBY

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

Annual Congress, April 11, 12 and 13, 1929

H. DICKINSON, *Reporter*

MR. CHARLES HOWARD USHER, *President, in the Chair*

FORMATIVE FIBROUS TISSUE REACTION IN THE EYE. MR. E. TREACHER COLLINS.

Mr. Collins described the form of reaction in the tissue of the eye which resulted in a new formation of fibrous tissue. He classified the conditions giving rise to this reaction as follows: (1) Encapsulating fibrosis. The first of the conditions under this head was that in connection with foreign bodies; the second in connection with neoplasms, and the third, with parasitic cysts. (2) Postfibrosis in connection with blood clots. (3) Reparative fibrosis: (*a*) in connection with developmental defects; (*b*) in the healing of wounds, and (*c*) as a sequel of granulomatous inflammation.

In order to explain the conditions observed, Mr. Collins formulated the following general propositions: 1. A formative fibrous tissue reaction was one of the means which the tissues of the body used for counteracting deleterious influences in their environment. 2. Such a formative fibrous tissue reaction might be excited apart from any exudation of leukocytes, though at times associated with it. 3. An aseptic foreign substance implanted in the body, exciting no exudation of leukocytes, might give rise to a formation of fibrous tissue which encapsuled and fixed it. 4. All neoplasms when they came into contact with tissue of mesoblastic origin excited a formative fibrous tissue reaction, this tending to check their extension into surrounding parts. In slowly growing neoplasms it succeeded in encapsulating the new growth. In the case of rapidly growing neoplasms, the cell proliferation outpaced the formative fibrous tissue reaction, and the growth took on malignant characteristics. 5. Any remedial treatment for neoplasms should aim not only at checking the proliferative activity of the cells composing it, but at promoting the formative fibrous tissue reaction. 6. Parasitic cysts coming into contact with tissue of mesoblastic origin excited a formative fibrous tissue reaction, an encapsulation in a fibrous tissue covering. 7. Coagulated blood in some situations in the body might act like a foreign substance and excite a formative fibrous tissue reaction. 8. A formative fibrous tissue reaction might be excited in the repair of lesions in the tissues of the body, due to defective development, traumatism or a destructive inflammation. 9. The new formation of fibrous tissue in the conjunctiva in trachoma was a secondary and reparative process which persisted, tending to replace the fibro-adenoid layer which was destroyed. 10. The new formation of fibrous tissue in the conjunctiva in spring catarrh was a secondary reaction excited by the proliferation and downgrowth of the epithelium, and it entirely disappeared when the disease came to an end.

DISCUSSION

SIR JOHN PARSONS: All these reactions of fibrous tissue can be regarded as reactions to irritation of mesoblastic tissue. If there is no mesoblastic tissue, the formation of fibrous tissue will not occur. The best example is in the vitreous, when there are vitreous hemorrhages. The ease with which vitreous hemorrhages are absorbed without the development of retinitis proliferans is astonishing, and is attributed to the absence of mesoblastic tissue. If the process is repeated, or if some toxic element is present, one finds that retinitis proliferans results. When it results, the cells are obviously derived from the mesoblastic tissue within the eye, i. e., from the mesoblastic tissue surrounding the blood vessels.

GRADED SQUINT OPERATIONS. MR. F. A. WILLIAMSON-NOBLE.

Mr. Williamson-Noble described modifications of the classic operations for squint, remarking that every operation for squint was a graded one. In place of tenotomy it was much simpler to put the muscle back a certain number of millimeters. He formerly used the Maddox tangent scale or the Priestley Smith tape, but there was a better method, consisting in the use of the Maddox rod and prisms graduated according to their angle of deviation. He performed the operation with the patient under general anesthesia, as it was pleasanter for the patient, and the operator could proceed with greater deliberation than with local anesthesia. He inserted the scleral sutures first. All but two of sixteen operations, which could be traced, were successful. One of the two failures was due to an error of judgment, as he underestimated the effect of Wilkinson's resection advancement. The squint was of 28 degrees, and the eye was slightly divergent; later it was 8 degrees and had not increased. The other was scarcely a failure, as that patient had a convergent squint of 45 degrees before operation, and it was reduced to 28 degrees. If consent could be obtained to operate on the other eye, it could probably be put straight.

SOME UNSOLVED PROBLEMS IN CONNECTION WITH DISEASES OF THE CHOROID. MR. MALCOLM L. HEPBURN.

Mr. Hepburn said that many investigators regarded the pathology of primary pigmentary degeneration of the retina as having been practically solved, but recently the controversy had ranged round two theories; one was vaguely described as nerve degeneration (presumably an ascending atrophy), the other, the vascular theory. Ten years before Mr. Treacher Collins brought forward a definite nerve theory, which that gentleman styled "abiotrophy of the neuro-epithelium." Mr. Hepburn challenged this and the other nerve hypotheses, and expressed his support of the vascular theory, the reasons for which he advanced with elaborate detail, and showed slides in confirmation.

Another problem still without solution was the actual reason for the variations in the defects of the visual field found as a result of inflammatory deposits and scars in the choroid. Two forms of scotoma occurred in such cases, one being the isolated islands corresponding to the size and shape of the deposit or scar; the other not only corresponding to the site of the original deposit, but extending out into the extreme limits of the peripheral field in the shape of a wedgelike defect, and involving, in addition, an apparently normal part of the retina. It had been too readily assumed that the wedge-shaped scotomas were due to pressure on the nerve fibers and the consequent loss of function in the peripheral parts of the retina from which these nerves were derived. If this was the only explanation,

one would have expected that the scotoma would follow the principles of a defect of a nerve fiber bundle, as seen typically in some cases of chronic glaucoma. Was it possible, he asked, that since isolated sectors of destroyed choriocapillaries had been demonstrated, there were also main arteries equally isolated and supplying definite regions, and thus an inflammatory deposit might block one of these main arterial channels and, in this manner, produce the sector-shaped defect in the visual field?

A further problem was why some diseases of the choroid terminated in secondary optic atrophy accompanied by diminution in the size of the retinal vessels, while in others, though the greater part of the choroid was involved, the disk and retinal vessels remained normal throughout. He thought that vascular diseases of the choroid were more likely to end in optic atrophy than were the inflammatory forms, and only those in which there was reason to believe that the lumen of the arteries was constricted by thickening of the coats of the vessel walls, thus interfering eventually with normal healthy nutrition of the whole neuro-epithelial layer.

Still another problem concerned the significance to be attached to the variations in amount and distribution of the pigment in diseases of the choroid, and what circumstances controlled the amount visible to the ophthalmoscopist. Also, did its appearance bear a relation to diagnosis and prognosis?

Thursday Afternoon Session, April 11, 1929

The Thursday afternoon session was devoted to a debate on heterophoria.

DR. E. MADDOX (Bournemouth): My aim is not to cover the whole subject, but only to touch on some interesting points. Orthophoria, from which heterophoria is a departure, can be defined as "a tendency for the eyes to set themselves truly for the object of fixation." Heterophoria can well be compared to the slackness in the steering-gear of an old motor car, which to a moderate degree is not consequential, but in larger amount makes too great a demand on the subconscious attention of the driver. It is a corresponding tax on attention which gives importance to heterophoria. Concentration of attention implies active inhibition of every conflicting neuron. Sherrington's "reciprocal innervation" in the motor sphere finding its counterpart in the psychic. The discomfort arising from heterophoria is apt to hover about the occipital region, whereas that of refractive errors is felt at the back of the eyes and in the temple and the brow. Sometimes a wandering eye suppresses its vision instantaneously, so that no diplopia appears; but, even so, the suppression makes no difference to the effect of the wandering on the aim of the other eye. In one well marked case, good binocular vision was enjoyed until the object came within 2 or 3 feet (60.96 or 91.44 cm.); then the right eye instantaneously suspended its vision and deviated without diplopia. Higher heterophoria is always concomitant and has to do with conjugate innervations, while lower heterophoria is always inconcomitant and has to do with the muscles or the terminal motor neurons. There are twelve species of paretic heterophoria and twelve of concomitant. The measurement for the paretic patient is on the same principles as that for frank paralysis. It is important to keep the disk of rods perpendicular to the line between the eye and the light; therefore, I have strung one on a kind of gallows, so that it might remain vertical however much askew the head might be placed. The concomitant heterophorias are due to slackness of one of the higher conjugate innervations. Exophoria for distance is nearly always due to either subsidence of muscle tone or convergence deficit.

It is rarely due to divergence excess. Division of the restiform body is said to cause conjugate version of the eyes to the opposite side, combined with elevation of one and depression of the other, confirming the see-saw innervations which I have described in a case. Cyclophoria, if constant, is due to relative inaction of either in cyclovergence (in which the two upper poles of the eyeballs approach one another) or in excyclovergence (in which they separate).

Dr. F. H. Verhoeff, of Boston, does not believe in cyclophoria, but believes that the fusion of skew images with one another is effected only by psychic means.

That is true within certain limits, but many tests show that the eyeballs can rotate about their visual lines. I should like to see a case in which, in nystagmus, the eyeballs cycloverged in and out, as that would afford a further confirmation of the innervations I am referring to.

The version phorias are less easily measured. Dextrophoria and levophoria are due to imbalance of two innervations; in these conditions there is a tendency for the eyes to look parallelwise to the right or to the left when at rest. Anaphoria and cataphoria mean a tendency for both eyes to look upward or downward, and are due to imbalance of the innervations, which I call surversion and deorversion.

Students are often perplexed between the terms "duction," "version" and "vergence." Whenever an eye is thought of monocularly one can speak of duction; it means that an eye is led by its muscles, and the mind does not travel beyond the orbit. Version is a conjugate parallel motion of the eyes effected by conjugate innervations. Vergences are contrary reciprocal motions effected by conjugate innervations. Hence when the amplitude of fusion is measured by prisms, it is better to speak of prism vergence tests than of duction tests.

Since no refraction is complete without testing for heterophoria, it is desirable to have a speedy method to single out cases requiring more accurate measurement. I have found that it is convenient to have a hand trial frame which can be held in front of the ordinary trial frame, the former having rods on one side and a rotating prism or pair of prisms on the other. The prism can be rotated after telling the patient to say "snap" when the red streak and the light meet. For accurate measurement a tangent scale is best, and if there is any suggestion that fusion is not completely suspended, one eye can be screened and then suddenly exposed, to see where the streak appears at the first moment of exposure. I regard the wing test as better, as it does away with the prism and ensures accommodation at the required distance.

Exophoria shown by the wing test, is generally of trivial account, but esophoria calls for a spherical correction which reduces it to nil. To give a weak prism for a marked exophoria is often of great value, as it is the last degree of stretch of the fusion reflex which is the most fatiguing. An approximate rule is, lenses for esophoria, prisms for exophoria.

It is impossible to make rigid rules as to the treatment for the condition, as it is a symptom, not a disease. The person must be treated, not the phenomenon. Most minor heterophorias do not need treatment, though exceptions can be admitted in the cases of gamesters, aviators and sportsmen, as it may have importance when the nervous system is tired. The counsel of perfection is to ascertain the cause and, if possible, to remove it. Errors of refraction are by far the most common cause, but septic foci in the nose, teeth or tonsils, lack of fresh air and exercise and anemia cannot be left out of account. If refraction treatment fails, the choice lies between exercises or relieving prisms. The former are more suitable for young persons, prisms for old. Any neurasthenia should be treated first. The effective value of a prism is less at a reading distance than for a far object. Also, one must make sure that the hyperphoria is not an after-effect

of wearing spectacles askew. If the condition is present only when the spectacles are on, the fault lies with the glasses, if vice versa the fault which has been in the spectacles is transferred to the eyes. The patient may have skewed his spectacles to correct the hyperphoria. For esophoria, prisms are less useful than lenses; the latter are curative. In exophoria, constitutional treatment is more often needed than in the other varieties; it is also the most suitable for training, especially when subconvergence co-exists. Weak prisms are often helpful; strong ones cause metamorphopsia, robbing the accommodation of the support it is accustomed to receive from convergence. Operations are suitable in selected cases of heterophoria; in neurotic people their effect is not permanent.

MR. C. GOULDEN: A routine examination of the muscle balance of patients who come with apparent or real refractive errors is obviously necessary. It is important to be certain of the type of deviation, i. e., whether it is concomitant or paralytic. The most important heterophoria is hyperphoria, a want of muscle balance between the eyes in the vertical direction; this form causes most inconvenience, because one has little power over the vertical deviation of the eye. Only when a vertical deviation causes symptoms should it be treated. Among the symptoms are persistent headache, often occipital, confusion of print and sometimes a complaint that the lower lines of print surmount the upper lines. The treatment consists in the use of prisms. If a low degree prism is necessary, this should be placed with the base down before the eye which tends to deviate upward. If the prism needed is 2 degrees or more, it should be divided equally between the two eyes; i. e., one prism base down before the upward turning eye, the other base upward before the downward turning eye. When of high degree, hyperphoria may call for operation; then advancement of either superior or inferior rectus should be done. I have seen four cases of torticollis due to hyperphoria.

There are patients who have a spasm of convergence immediately the examination is commenced; these are probably hysterical in origin, and need a careful correction of refraction, probably with the use of atropine, and then such treatment as the hysteria may call for. I have neither operated nor supplied prisms for this condition. In patients who resist treatment by exercise, especially those in whom an exophoria becomes a squint, i. e., when the patient is fatigued, operation may be necessary. Here, tenotomy is not usually regarded as sufficient; there must be advancement of an internal rectus muscle.

GROUP-CAPTAIN CLEMENTS (R.A.F.): In my experience with flying men, I can point out that in the case of an aeroplane in space the strains and stresses differ from those in persons on the ground. I shall analyze the results of spinning in the Barani chair ten times in twenty seconds in the case of 118 men. Practically all flying men have good vision. I believe that a large percentage of patients with torticollis will be found to be suffering from hyperphoria.

MR. ERNEST CLARKE: I do not think it is fair to take as normal the kind of case encountered in the air force. In the ordinary population, I regard hyperphoria as common, but as it is usually a minute degree of hyperphoria, it does not matter. For many years I have advocated the correction of low errors of refraction. When minus glasses are necessary, there may be a small exophoria. The latter always lowers the amplitude of convergence; when exophoria is present, the fusion supplement required is always greater, whereas the ophthalmic surgeon should make the fusion supplement as low as possible. When low refractive errors are corrected, the hyperphoria usually disappears in a short time. One should not be content with a single test. I object to the use of prisms for young or middle-aged persons; to supply these at this stage is a confession of weakness, and the strength has to be increased progressively.

MR. H. GRIMSDALE: Patients are seen occasionally who are greatly helped by a cylinder of 0.25 diopter and by a prism of 1 degree. Can rules be laid down for dealing with the condition? Probably not. For exophoria I usually advise exercises. I do not think that the rôle of hyperphoria in the production of strabismus has been given sufficient attention. Cyclophoria rarely gives trouble.

MR. HUMPHRY NEAME: Marked exophoria is found occasionally with the Maddox rod test. In such cases, by taking a red green light, a red green glass and a prism to dissociate the two eyes, exophoria is found to be definitely less. I agree in regard to the use of a prism to relieve discomfort due to hyperphoria, and the remark that the smallest possible prism should be used.

I believe that it is wrong to prescribe a prism of any magnitude to any except aged persons.

MR. T. HARRISON BUTLER: If one orders prisms, especially for convergence, it is essential to see the patient more frequently than usual, as it is my experience that often, when ordering prisms for elderly persons with a convergence insufficiency, the relief given tends to allow the muscles to repair, and then the prisms can be discarded.

MR. F. A. WILLIAMSON-NOBLE: It is stated that there are no means of relieving cyclophoria, but I do not agree. It depends on the following principle. If one takes a mirror and holds it in front of the eye at 45 degrees, one sees a reflection of what is behind it. If the mirror is tilted one sees a lateral tilting in the image. An apparatus has been made in America in which a mirror is placed in front of the eye, and another placed behind, so that the image is straight ahead, and the deviation from the tilting mirror is compensated by the prism. By adjusting the mirrors one can get 4 or 5 degrees of tilting in the image being looked at with the cyclophoric eye.

DR. G. MACKAY: As long as the degree is low, prisms can be tolerated, but in high degree the necessary prisms cause great inconvenience. I have been struck with the variability of hyperphoria, even from moment to moment, in the course of an examination. I have operated freely on the superior or inferior rectus for from 10 to 15 degrees, and I have not found it necessary to do advancement.

Friday Morning Session, April 12, 1929

THE STRUCTURE OF THE VITREOUS BODY. MR. W. S. DUKE-ELDER.

The investigation of the structure of the vitreous body was described as revealed by the ultramicroscope, which provided magnifications up to colloidal dimensions, and necessitated no fixatives before its use, allowing fresh and untouched material to be studied directly. With this instrument it could be shown that the vitreous was a pure homogeneous gel, that the hyaloid membrane, in the accepted sense of the term, did not exist, and that the zonule had a structure similar to that of the vitreous. The gel was made up of colloidal particles, or micellae, of fibrillar shape, similar to those seen in soap solutions. This formed the basis for the so-called structure, which could be seen histologically or with the slit-lamp. Study of the vapor pressure isotherm of the vitreous and its behavior on filtration showed that it was an unstable gel, liable to get broken down by slight mechanical trauma, and that it was elastic, turgescible and hydrophylic. In chemical constitution, it was compared with the aqueous humor and the blood plasm, and it was shown that its composition could be explained

in physicochemical terms as being identical with the aqueous humor, with the addition of mucoprotein for maintaining optical transparency and a residual protein, the function of which was gelability. He suggested that these two constituents, each with a special function, were secreted from the ectodermal tissues surrounding the vitreous chamber, these being percolated by the common intra-ocular fluid, which was a dialysate capillary blood. The result was a swollen mass in the form of a gel. If the gel swelled, glaucoma was produced, if it shrank there was a liability to detachment of the retina. If the gel broke down, the vitreous liquefied, and the deposition of a coagel resulted in opacities. The influence of variations in the volume of the lens and the sclerotic was seen to control the final intra-ocular pressure. Experiments were described illustrating the variations in tension produced by perfusion of the blood vessels of a "surviving eye preparation" with blood, showing changes in its phosphorus and salt content. It was concluded that with an increase in alkalinity and a decrease in salt concentration the tension of the eye should rise; it was suggested, however, that the more important factor in the changes of the intra-ocular pressure which occurred clinically would be found in alterations in the condition and chemical relationship of the constituents of the vitreous itself under pathologic conditions. To this, he said, research was now being directed. It might be possible to treat glaucoma by inducing shrinkage of the gel by increasing its hydrogen ion concentration by iontophoresis or other means; to treat detached retina by causing swelling in the gel, and to treat a fluid vitreous by bringing about a solgel transformation.

THE NATURE OF TAY'S CHOROIDITIS AND ALLIED CONDITIONS. MR. DAVENPORT.

Mr Davenport suggested a classification, somewhat on the following lines: (1) A main group of colloid body formation, (*a*) juvenile or congenital, (*b*) senile, i.e., first seen in old people, (*c*) associated with gross fundic changes. (2) Changes simulating colloid body formation in the clinical picture presented. In all younger patients, he said, the changes were mostly of the widespread type, but in none of them was vision impaired. Many of the casts of massive exudation showed discrete spots. Most of the cases had been called Tay's choroiditis. A good prognosis should not be given as to vision in a case looking like colloid body formation in which the other eye showed grosser changes, or in which vision was affected in the eye with the apparent colloid body formation. Nettleship classed Tay's choroiditis with senile changes in the macula.

DISCUSSION

MR. E. TREACHER COLLINS: In the cases which the late Mr. Doyne described as "honeycomb choroiditis," was there a hereditary disease which could often be traced in several members of the same family or in several generations? I published a description of sections of such a case. In the macula region, in which there was a white area, I found hyaline material similar to that composing nodules in so-called Tay's choroiditis; the spots were on the inner surface of the lamina cribrosa. There was no change in the choroid or in the vessels themselves. The best name for it is a giant Tay condition.

MR. HUMPHRY NEAME: I have thought for a considerable time that cases diagnosed as Tay's choroiditis in which the vision was affected to some extent were due simply to colloid bodies pushing into the retina and so damaging the rods and cones that central vision was affected.

THE PHYLOGENETIC VALUE OF THE LOWER RETINAL FIELD. MISS IDA MANN.

Miss Mann showed that there was a big difference between the upper and lower retinal areas. The lower retinal field could be regarded as fluid, the upper part as stable. Throughout the whole scale of the embryo the lower retinal field was younger than the upper. She thought the explanation must be that the lower retinal field, which provided the upper part of the field of vision, was not of such great importance as was the upper retinal field; it was more essential for an animal to see things on the ground and directly in front of it than to see things which were very much over its head.

THE GREAT ANTIQUITY OF REMOVAL OF CATARACT FROM THE EYE. MR G. F. ALEXANDER.

Mr. Alexander gave suggestions on the instrumentation of cataract operations, showing a number of modifications in instruments. He was able to show that removal of cataract from the eye was carried out as early as 2000 B. C.

THE PATHOLOGY OF ORBITAL HEMORRHAGE AND INFLAMMATION. MR. E. WOLFF.

Mr. Wolff showed that the reason a hemorrhage into the orbit took a longer time to absorb than one in most other parts of the body was largely that the orbit, especially after the accompanying edema, was a closed cavity, similar to the skull. The only direction in which the contents of the orbit could expand was anteriorly; hence swelling of the tissues caused a proptosis. It also explained the recovery in cases of orbital inflammation after a simple incision into orbital tissues. The rapid onset of blindness noted in many patients with orbital cellulitis was, no doubt, due to pressure exerted on the optic nerve itself or on its blood supply. He quoted cases.

Friday Afternoon Session and Friday Evening Session, April 12, 1929

On Friday afternoon, a number of clinical cases were exhibited and discussed at the Royal Westminster Ophthalmic Hospital. The evening of that day was devoted to a discussion on the diagnosis and treatment for ocular tuberculosis.

SIR ARNOLD LAWSON: The diagnosis rests largely on the presence of active tuberculosis elsewhere in the body. In the eye the disease is essentially surgical, as opposed to medical. The fact that there is no evidence of active tuberculosis elsewhere is not evidence that in the particular case one is not dealing with surgical tuberculosis. Ocular tuberculosis in relation to pulmonary phthisis is, however, uncommon. In children, even up to puberty, bovine, as opposed to human, tuberculosis is fairly frequent, as shown in adenitis, infected glands and enlarged tonsils; some show no other physical signs. A negative result with tuberculin from human beings does not eliminate bovine tuberculosis. The co-existence of tuberculosis with congenital syphilis is common in young persons, and there is no reason why oral sepsis should not co-exist with active tuberculosis. Tuberculous lesions exhibit a marked tendency to relapse when healing is apparently complete. There is a remarkable absence of severe pain in these cases. I consider Calmette's local conjunctival test too dangerous to use; if it were used, a focal reaction in the eye would be diagnostic.

Tuberculous diseases of the eye may be extra-ocular or intra-ocular. The first is of two kinds, so-called primary and secondary; however, I do not think that tuberculosis of the eye, even of the conjunctiva, is ever primary. Intra-ocular tuberculosis may occur in any part of the uveal tract. Iridocyclitis is the most common of all intra-ocular tuberculous infections or inflammations, but frequently the evidence of tuberculosis is absent, and there is little to show beyond the history and the clinical course.

The treatment for ocular tuberculosis must be considered from the general and the local points of view. In a large proportion of children enlarged tonsils and adenoids must be dealt with before treatment of the eye condition can be expected to respond well. There is much difference of opinion as to the value of treatment by tuberculin. Von Hippel reported that in 243 patients with ocular tuberculosis treated with tuberculin, 75 per cent were cured, only 7 per cent showing no improvement. Sir Arnold said that it was necessary to make sure that the patient in a given case was suitable for the treatment before using it. I believe that the value of bovine tuberculin has not been fully appreciated by the medical profession. Much good has been done for these patients in this country by Goulden and Duke-Elder particularly, who administered phototherapeutic measures. I regard the value of local light therapy in these cases as still sub judice.

DR. R. A. YOUNG: I deal with tuberculosis from the general systemic standpoint.

MR. S. BROWNING: I am a firm believer in tuberculin therapy for ocular tuberculosis; however, the type of disease must be determined so that one can select the suitable tuberculin for the treatment. I have been using both tuberculin from human beings and bovine tuberculin for fifteen years.

MR. A. L. WHITEHEAD (Leeds): Supplementary general constitutional treatment of patients suffering from ocular tuberculosis is important. I give tuberculin tentatively at first, the beginning dose being usually 0.005 or 0.0001 mg. according to the age of the patient.

MR. W. S. DUKE-ELDER: In thirty-three patients with ocular tuberculosis, in whom the duration of treatment before the use of ultraviolet light was two and one-half years, and in whom the latter treatment was used after five months —75 per cent of whom also received injections of tuberculin — 85 per cent showed quiet eyes, 6 per cent much improvement and only 9 per cent still had active disease.

Saturday Session, April 13, 1929

OPTIC ATROPHY FOLLOWING HEMORRHAGE FROM THE ALIMENTARY TRACT. MR. MAURICE WHITING.

Mr. Whiting said that the occurrence of a condition resembling optic neuritis and followed by optic atrophy had long been a well recognized complication of some cases of severe hemorrhage from the alimentary tract and elsewhere in the body. The author described three cases which he had encountered. The changes in the fundus reported in these cases were of varying character, from pallor and slight blurring of the disk to an apparently definite optic neuritis, with hemorrhages and exudates. The question arose as to whether or not the condition of the fundus was that of a definite optic neuritis. His own view was that the first state was not optic neuritis necessarily, but might be due to a sudden death of the nerve fibers, as none of the signs of a secondary atrophy were seen in the final condition.

THE PREVENTION OF MINOR INJURIES OF THE EYE IN INDUSTRY. MR. B. CRIDLAND.

Mr. Cridland dealt with the subject in an exhaustive manner. In 70 per cent of cases no protection for the eyes was available or used, showing neglect on the part of employers; in 19 per cent protection was provided but was not made use of. In 11 per cent of the accidents to the eyes during occupation protection, chiefly in the form of goggles, was used, so that the protection was insufficient. Goggles which had been damaged during use were shown. Exhibits by Mr. Cridland were shown in the museum.

During the three days of the congress, a museum was run in the building, at which a number of well known optical firms exhibited.

Book Reviews

BIOMICROSCOPIE DE LA CHAMBRE ANTERIEURE DE L'IRIS ET DU CORPS CILIAIRE. JACQUES MAWAS, M.D. Société française d'ophthalmologie. Price 120 francs. Pp. 116 with 30 plates. Paris: Masson & Cie, 1928.

The first part of the Microscopie de l'oeil vivant, which the French Ophthalmological Society is bringing out, appeared in 1926. It treated of the diseases of the cornea, and was written by Prof. E. Gallemaerts, of Brussels. This is the second part, and as the author, Dr. Mawas, states in his preface, it differs from the usual "rapport" in as much as there is more personal labor than a study of the bibliography, and each drawing represents an original observation. His object has been to show how this new technic can serve early diagnosis, and makes for a better comprehension of the pathologic processes at work, which is in short the entire worth of biomicroscopy. After an historical introduction of the slit lamp, which instrument the author places in the same importance as the ophthalmoscope, he takes up the important relations between biomicroscopy and normal and pathologic histology, stating that biomicroscopy is still in the purely descriptive anatomic phase. This is especially true for the lens where many forms of cataract have been described without a word on the true nature of the lens. Nor have the observations of the slit lamp been completed by those of normal or pathologic histology. This omission has been corrected in this atlas whenever possible. Biomicroscopy is really histology without fixation or staining and acquires its full value only when controlled and completed by the histology of the laboratory. Thus it presents a method of approach which still is practically unexplored. The technic is not difficult, but a preliminary knowledge of the normal histology is essential.

The study of the anterior chamber and its contents the aqueous humor, of the lens and of the vitreous is preceded by the description of their physical and chemical structure. In brief, the eye is an organ containing three colloids which correspond to three transparent parts. Biomicroscopy permits a study of the fundamental processes of this new chapter in physicochemical ophthalmoscopy.

Part 1 deals with biomicroscopy of the anterior chamber. The aqueous is not absolutely transparent but its opalescence is proportionate to the amount of protein contained in a colloidal solution. To examine the aqueous, it is necessary to employ a narrow slit or a stenopaic hole and an intense light, remembering to focus the light bundle back of Descemet. Directions are given for measuring the depth of the chamber. The contents of the normal aqueous are then studied: its living elements, the Ehrlich-Türck line, convection currents. The pathologic aqueous which corresponds to the second aqueous is readily illuminated and is described as cloudy or granular with fibrin, or containing cellular elements, corneal deposits and pigment. The author explains the localization of deposits on the posterior surface of the cornea by convection currents, superficial tension, the electric state of the wall and the ionic concentration of the aqueous. A chapter is devoted to gonioscopy giving

the methods of studying the angle of the anterior chamber as suggested by Koeppe and Troncoso. The ultramicroscope, spectroscope and microscope with polarized light are briefly mentioned.

Part 2 deals with the iris. The author believes that the biomicroscope can show in the iris microscopic lesions in the stroma and in the pigment epithelium of the retina. The iris should be regarded as a picture of the choroid exposed to view. The various methods of illumination to show the iris lesions are then taken up. Normal histology of this iris as described by Borel and P. Masson follow, and the author speaks of a trophomelanotic network (cells in the anterior and posterior mesodermal layers). Biomicroscopy of the iris is daily bringing out discoveries both in the normal and in the pathologic. The color and structure of the iris, congenital anomalies, senile changes, traumatic lesions, tumors including cysts, and diseases of the iris, the formation of nodules, atrophies, are all taken up in turn.

The author speaks of an iritis of the posterior epithelium in which he regards the depigmentation as the important lesion.

Part 3 deals with the biomicroscopy of the ciliary body and of the zonule. This examination is possible only in the presence of an iridectomy, iridodialysis or aniridia.

A bibliography and numerous excellent plates, many in color, conclude the volume.

The author has presented his subject in an interestingly broad and original way based on the fundamental teachings of physiology and histology. Its careful study can be recommended, and the reader will be repaid by many valuable suggestions and lines for new investigations.

A. K.

PHOTOGRAPHS OF THE FUNDUS OCULI. By ARTHUR J. BEDELL, M.D. 95 plates, 324 single pictures, 272 stereoscopic photographs. Philadelphia: F. A. Davis Company, 1929.

The author believes that with photographs of the fundus oculi, a better conception of changes in the blood vessels, optic nerve, retinal and choroidal lesions is possible, and that the illustration is rather enhanced by absence of color. This atlas, the first in English, is based on 4,000 photographs made with the Nordenson camera of Zeiss. The pictures are grouped to display the normal fundus with its physiologic variations, the lesions of the retina, choroid and optic nerve, and in terms of general subjects like arteriosclerosis, syphilitic disease, diabetes and others. In the foreword, the author speaks of the necessity of first familiarizing oneself with the characteristics of photographs of the fundus; thus only can the various color densities be appreciated. The refractive error must be remembered in the case of the transitions of the fundus. Changes in the nervehead and in the course of the vessels are then described. The macular region comes next in the examination, and the author believes it is best studied by photography. Then follow variations of pigment in the fundus, the identity of a vessel, of an exudate, syphilitic organic changes as they occur in chorioretinitis, in the vessels and optic nerve, senile changes, holes in the macula, tuberculosis, choked disk, optic atrophy, cupping of the optic disk, arteriosclerotic changes, chronic interstitial nephritis, retinal hemorrhages and diabetic retinitis.

Facing each picture is a description of the fundus change and a few clinical notes of the case depicted.

The photographs measure only 1⅜ inches. They are small but unusually distinct and are improved with a magnifying glass. While they convey much information for the practiced eye, they are undoubtedly difficult for the beginner in ophthalmoscopy. The stereoscopic plates when properly examined reveal a remarkable degree of plasticity.

The author is to be congratulated on this remarkable achievement; one cannot but wonder at the industry and enthusiasm which this work called for, and the strikingly successful reproduction of the photographs deserves special commendation.

This is only the beginning of the application of photography to clinical ophthalmology. How much photography will replace and supplement the present written descriptions of fundus lesions, and its practicability in every day practice are interesting speculations.

A. K.

SPONTANEOUS CURES OF HOPELESS DISEASES. By DR. CARL HAMBURGER. Pp. 41. Jena: Gustav Fischer, 1928.

Spontaneous cures, being nature's technic of curing, are of immense theoretical and practical interest. Hamburger's idea of collecting from the ophthalmic literature and from his personal experience "hopeless" cases in which the conditions have healed spontaneously is a felicitous one. Of the cases reported, the following are worthy of mention: three cases of sympathetic ophthalmia in which the condition disappeared during an attack of an acute febrile disease; one case of severe tuberculosis of the cornea and conjunctiva which cleared up after an attack of erysipelas of the face; another case of the same type in which the condition disappeared after a severe tuberculin anaphylaxis; a case of recurrent hemorrhages in the vitreous in which the condition was cleared up by repeated venesections; a few cases of severe trachoma with pannus which cleared up by an induced gonorrheic ophthalmia; one case of optic atrophy in which the patient recovered normal vision after an attack of smallpox; another case in which spontaneous recovery of useful vision obtained without the occurrence of an intercurrent disease; a few cases of senile cataracts in which the opacities liquefied and resorbed, and finally, a case of glaucoma simplex in which the eye softened after two attacks of acute iritis.

It is questionable whether the cases of corneal tuberculosis which cleared up after a severe tuberculin anaphylaxis or those of trachoma which cleared up after conjunctival inoculation of gonococci are real instances of spontaneous cures. The physician had done something in these cases to start nature's healing process.

According to Hamburger, the common factor that had something to do with the spontaneous cure was an acute febrile disease, but his logic is not entirely unassailable. For every case of such spontaneous cure, one can find ten others in which the conditions are not cleared up after an intercurrent febrile disease.

The author's method of presentation is stimulating, and though nothing in this book is original and compelling, its merit lies in reminding one that pessimism has no place in medicine. Let one continue to watch for the slightest hint of nature's ways of curing "hopeless diseases."

MARK J. SCHOENBERG.

A Text Book of Eye, Ear, Nose and Throat Nursing. By Abby Helen Denison, R.N. Price, $3. Pp. 295. New York: The Macmillan Company, 1929.

This is a textbook for nurses, growing out of teaching at the Massachusetts Eye and Ear Infirmary. It begins with an elementary anatomy of the eye, then follows a chapter on drugs and solutions, the procedure of eye treatments, with the applications and treatments in general use. The common diseases of the eye are described with their special forms of treatment and their nursing care. A chapter is devoted to the preparation of a patient for an operation on the eye, and the postoperative care. The technic of the operating room, and of the outpatient department completes the part on the eye.

This is undoubtedly a useful book, and a great deal of information has been assembled. It cannot, of course, replace practical experience, for in nursing of the sick there are many facts which never find their way into print.

A. K.

Lehre von den Gesichtsempfindungen. Publications of the late Franz Hillebrand, collected and edited by his widow Franziska Hillebrand, M.D. Price, 14 marks. Pp. 205, with 40 text illustrations. Berlin: Julius Springer, 1928.

The author's painstaking labors over a period of thirty years in the field of visual impressions are now collected and edited by his widow, who was first his pupil, and then became his associate in many of his investigations. Dr. Hillebrand was a pupil, then a disciple of Hering, and has now made accessible the investigations of that great psychologic physiologist. These embrace studies on the light sense and the space sense. The former gives the dependence of color perception on stimulation and on excitation and reviews the color theories. The latter deals with the space sense in general, localization by the eye at rest and in movement, and finally a discussion on nativism and empiricism. This scholarly treatise is of the greatest importance to workers in this abstruse field.

A. K.

Directory of Ophthalmologic and Otolaryngologic Societies *

FOREIGN

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. A. B. Cridland, Salisbury House, Chapel Ash, Wolverhampton. England.
Secretary: Dr. C. G. Russ-Wood, 12 St. John's Hill, Shrewsbury, England.

INTERNATIONAL

INTERNATIONAL CONGRESS OF OPHTHALMOLOGY

President: Dr. E. Treacher Collins, 17 Queen Anne St., London, England.
Secretary: Dr. E. Mark, Academisch Zickenhuis, Leyden, The Netherlands.
Place: Amsterdam, The Netherlands. Time: Sept. 5-13, 1929.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

Chairman: Dr. T. B. Holloway, 1819 Chestnut St., Philadelphia.
Secretary: Dr. William C. Finnoff, Imperial Bldg., Denver.
Place: Detroit. Time, 1930.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Harris P. Mosher, 828 Beacon St., Boston.
President-Elect: Dr. William H. Wilder, 122 S. Michigan Ave., Chicago.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: Atlantic City, N. J. Time: Oct. 21-25, 1929.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Campbell Posey, Radnor, Pa.
Secretary: Dr. Emory Hill, 501 E. Franklin St., Richmond, Va.
Place: Richmond, Va. Time: June, 1930.

LOCAL

LOS ANGELES COUNTY MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Harold S. Muckleston, 6422 Hollywood Blvd., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 1208 Roosevelt Bldg., Los Angeles
Place: California Lutheran Hospital, Assembly Room. Time: 8 p. m., first Monday of each month.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Chairman: Dr. Louis S. Greene, 1710 Rhode Island Ave. N. W., Washington.
Secretary: Dr. R. E. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

ATLANTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. F. McDuffie, 45 Edgewood Ave., Atlanta, Ga.
Secretary: Dr. Leon E. Brawner, 157 Forrest Ave. N. E., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. N. E. Time: 8 p. m., last Thursday of each month.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Robert J. Masters, Hume-Mansur Bldg., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. M. Earle Brown, Maison Blanche, New Orleans.
Secretary-Treasurer: Dr. W. A. Wagner, Pere Marquette Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., third Thursday of each month from September to June.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Leo J. Goldbach, 1012 N. Charles St., Baltimore.
Secretary: Dr. Angus Lloyd MacLean, Johns Hopkins Hospital, Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Holbrook Lowell, 82 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. E. B. Dunphy, 520 Commonwealth Ave., Boston.
Place: Massachusetts Eye and Eye Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

DETROIT OPHTHALMOLOGICAL CLUB

Chairman: Members rotate alphabetically.
Secretary: Parker Heath, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. R. Forgrave, Logan Bldg., St. Joseph, Mo.
Secretary: Dr. A. E. Eubank, Argyle Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. William H. Luedde, Metropolitan Bldg., St. Louis.
Secretary: Dr. Leo L. Mayer, Carleton Bldg., St. Louis.
Place: St. Louis or Washington University Cafeteria.
Time: 6:30 p. m., fourth Friday of each month from October to April, inclusive.

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Frederick J. Wort, 1080 Broad St., Newark.
Secretary: Dr. Earle LeRoy Wood, 31 Lincoln Park, Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. Robert M. Rogers, 680 St. John's Pl., Brooklyn.
Secretary: Dr. William F. C. Steinbugler, 515 Ocean Ave., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday of February, April, May, October and December.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. John P. O'Keefe, 251 State St., Albany.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.

BUFFALO OPHTHALMOLOGIC CLUB

President: Dr. E. E. Blaauw, 190 Ashland Ave., Buffalo.
Secretary-Treasurer: Dr. A. F. Luhr, Medical Arts Bldg., Buffalo.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. E. F. Krug, 12 W. 44th St., New York.
Secretary: William F. C. Steinbugler, 815 Park Ave., New York.
Time: 8:30 p. m., third Monday of every month.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Frank Barber, 75 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Cecil B. Hert, 29 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Assn., 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George M. Constans, Bismarck.
Secretary-Treasurer: Dr. F. L. Wicks, Valley City.

CLEVELAND ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Chairman: Myron Metzenbaum, Rose Bldg., Cleveland.
Secretary: Dr. A. D. Ruedemans, Euclid and E. 93d St., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Charles M. Swab, Medical Arts Bldg., Omaha.
Secretary: Dr. J. C. Davis, 1615 Howard St., Omaha.
Place: Medical Arts Tea Room. Time: 5:15 and 7:00 p. m. Third Wednesday of each month from October to May.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Joseph L. McCool, 909 Stevens Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

THE PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

THE PITTSBURGH SLIT-LAMP SOCIETY

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Arthur Jones, Boise, Idaho.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. C. E. G. Shannon, 17th and Walnut Sts., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. William Thornwall Davis, 927 Farragut Sq., Washington, D. C.
Secretary: Dr. Fletcher D. Woodward, Box 162, University, Va.
Place: Miami, Fla. Time: Nov. 20-22, 1929.

THE MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. D. L. Bettison, Medical Arts Bldg., Dallas, Texas.
Executive Secretary: Dr. W. Mood Knowles, Medical Arts Bldg., Dallas, Texas.
Recording Secretary: Dr. L. A. Nelson, 4105 Live Oak, Dallas, Texas.
Place: Dallas Athletic Club. Time: 6 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. P. Schenck, Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Needham, Fort Worth National Bank Bldg., Fort Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

HOUSTON ACADEMY OF MEDICINE, EYE EAR, NOSE AND THROAT SECTION

President: Dr. John H. Foster, 1300 Walker Ave., Houston, Texas.
Secretary: Dr. Edward W. Griffey, Houston Clinic, Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

THE SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. F. H. Roseborough, 302 Moore Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Will Otto Bell, Medical-Dental Bldg., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, Medical-Dental Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Herschel Ezell, Doctor's Bldg., Nashville, Tenn.
Secretary-Treasurer: Dr. W. W. Wilkerson, Jr., Bennie-Dillon Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. H. B. Stone, Roanoke.
Secretary-Treasurer: Dr. Fletcher D. Woodward, Box 162, University.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. O. M. Rott, Paulsen Bldg., Spokane Wash.
Secretary: Dr. C. A. Veasey, Jr., Spokane, Wash.

THE MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. L. P. Allen, 145½ Main St., Oshkosh, Wis.
Secretary-Treasurer: Dr. Edward R. Ryan, 425 E. Water St., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.